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ROENTGEN STUDY OF THE MUCOSA OF THE COLON*†

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BEFORE taking up the interpretation of the study of the mucosa of the colon, it is necessary to discuss briefly some points of technique, because in order to visualize the internal relief of the intestine, the examiner must use a very exact procedure. This depends on the preparation of the patient, on the opaque medium used, and on the method of the examination itself.

Preparation of the Patient. The intestine should be completely empty. For this castor oil as a purgative the night before (25 to 30 grams) is invaluable, followed the next morning two and one hours before examination, by two cleansing enemas of from 1 to 2 liters of tepid water. The shadows in an intestine which has not been completely emptied will lead to serious errors.

Choice of Opaque Medium. Many authors have obtained good images of the mucosa by using suspensions of barium, but often the shadows, particularly in pathologic cases, lack detail. The coating is insufficient and the diagnosis can be made with great difficulty.

Frick, Bluhbaum and Kalkbrenner (1928) have used a new product, a colloidal solution of thorium dioxide, which has the property of flocculating on the intestinal mucosa. This flocculation depends on the pH of the intestinal contents, and undergoes important variations so that it is necessary for one to have at his disposal solutions the flocculation of which is more or less definite. These solutions are commercially available under the names of collothor, diagnothorine or umbrathor, and have the advantage of producing a real coating on the intestine, in contrast to the sulphate of barium suspensions, which adhere to the mucosa only by capillarity. These solutions have a concentration of from 25 to 30 per cent, and should be diluted to from 6 to 12 per cent by distilled water.

Technique of Examination. The examination is divided into three stages: (1) filling with slight distention; (2) evacuation of all excess; (3) insufflation. Each step is followed by roentgenoscopy, but it is also necessary to make a roentgenogram at each step for verification of the findings.

(1) Filling with Slight Distention: Overdistention of the intestine must be avoided if one desires to obtain a good collapse of it. When the caliber of the intestine has been distended beyond a certain limit, the distention persists for some time due to a more or less marked paralysis. The intestinal wall is incapable of contracting and of expelling its contents. To fill the intestine without distending it, it is necessary to fill it in short stages and with firm pressure of

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Fig. 1. Roentgenogram of the rectosigmoid region, normal folds of the longitudinal type.

from 0.4 to 0.6 atmosphere. There is a special instrument to simplify this technique.

(2) Evacuation of All Excess, Study of the Collapse: The quantity injected is from 400 to 800 c.c. and the patient retains the opaque enema ten to fifteen minutes. Then the excess is evacuated without effort. It is important to control this evacuation, which should not be total or incomplete, or unequal. All these are details of technique into which it is impossible to enter here.

(3) Insufflation: This is done with the patient lying on his back, either with a thermocautery air bulb (the bulb of Richardson), or with the Küss instrument, modified by G. Maingot, which permits constant measurements of the quantity of air injected. The air should be injected gently so as not to cause the patient discomfort. The filling of all the segments is checked by roentgenoscopy (Fig. 3).

NORMAL MUCOSA

Since 1930, with my associate Kadrnka, I have been studying the mucosa of the colon at l'Institut Central de Radiologie de Genève, on the service of Professor Gilbert. Since 1933, we have been carrying on these studies at the Hopital Laennec in Paris, on the service on Dr. Maingot. There is in press a book, containing about 200 illustrations, which has been written in collaboration with Drs. Maingot and Duclos.

In order to interpret the image of the folds, a knowledge of the different normal forms of folding is necessary. The work of Forssell (1913) is the anatomical and physiologic basis for this study. To this fundamental work there has been added the studies of Adam, Albrecht, Berg, Chaoul, Cole, Dell'Acqua, Fischer, Fleischner, Gilbert, Kadrnka, Knothe, Ledoux-Lebard, Maingot, Schinz, Villardell, and others.

Simple Folding. We regard simple folding as that which is made up of a single type of fold, either longitudinal or transverse. Longitudinal folds are seen most often in the rectosigmoid and descending colon (Fig. 1). They are made up of a chain of three to five links at the most. These latter are not absolutely parallel and sometimes they anastomose with each other. Transverse folding is relatively rare, and is a series of rings dividing the intestine. It is exceptional that the whole intestine will show one type of folding which varies from one segment to another and from one time to another. All these images are transitory and depend on the function of the intestine and on its degree of collapse.

Composite Folding. This is due to the combination of longitudinal and transverse

Fig. 2. Roentgenogram after evacuation; normal mucosa, alternation of longitudinal transverse and oblique folds, air in the rectosigmoid.

folds. The longitudinal folds may be more or less long and in places they are interrupted by transverse folds which are generally grouped (Fig. 2).

One of the most frequent forms of composite folding is branched folding, due to a combination of longitudinal, transverse and oblique folds. At first sight they may seem to be incapable of interpretation, but with practice one may easily differentiate the normal from the pathologic. The normal has a symmetry, a regularity, which is not found in pathologic conditions. Another point which should not be neglected is the number and the caliber of the folds.

All these aspects of the normal mucosa require more extensive description, but we prefer to speak only of the results of the study of the folds. We will therefore discuss principally the study of certain syndromes. All our evidence is based on clinical control and anatomical and pathologic study. One should interpret the findings in the roentgenogram on the basis of anatomy and physiology. The arrangement of the folds is a representation of the condition of the internal surface of the intestine.

REACTIONARY STAGES

These are the first responses of the intestine to an irritant of any sort, whether of an abdominal or a general nature.

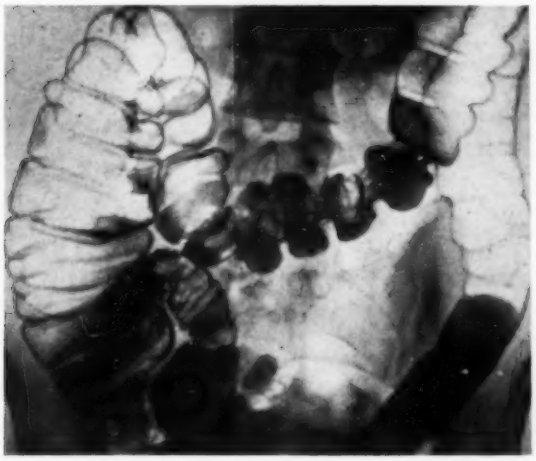

Fig. 3. Roentgenogram after insufflation; mucosa normal, homogeneous density, normal striping of the mucosa.

Fig. 4. Roentgenogram after evacuation; folds of the irritative type, curly and tufted.

Among the general causes there might be cited, nervous, endocrinal, humoral, alimentary, sanguine, those due to dyscrasias, to medicamental, toxic causes, etc. Among the abdominal causes are those of an intraperitoneal and extraperitoneal nature. In the latter group are included those of colic and extracolic origin.

These reactionary stages must be sharply differentiated from changes due to colitis, for they are the stages which precede colitis. It is possible to follow all the stages, from that of simple irritation to actual colitis. These reactionary stages cannot be recognized otherwise than by a study of the folds, which are so important that a special technique of examination may be necessary. In fact, when the intestine is distended by the opaque liquid it often happens that it is impossible to estimate exactly the degree of intestinal irritation, aside from the fact that the intestine has

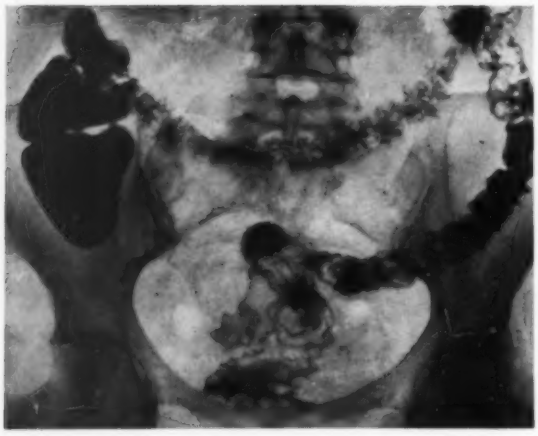

FIG. 5. Roentgenogram after evacuation; on the right border of the sigmoid, an opaque spot, joined to the colon by a fistulous tract, perisigmoid abscess, invisible after refilling.

a spastic appearance. In contrast to colitis, these stages might be called coloses. However, the termination "osis" implies a degenerative condition, one which is found only very exceptionally in these reactionary stages.

In this group two factors dominate the morphology of the folds, the one, neuromuscular, the other liquid. When the neuromuscular factors predominate there is an increase in the number of the folds, giving the appearance of colico-mucosa described by Knothe. This is the irritative stage (Fig. 4). On the other hand, when the liquid factor predominates, the submucosa is turgescent, the folds are wider, but the number is about normal. This is the exudative stage. The folding is therefore richer than normal, its caliber is widened, it has a loose appearance. This is the most marked stage of the reactionary phase and is the beginning of mucocatarrhal colitis.

MUCOCATARRHAL COLITIS

Mucocatarrhal colitis has a characteristic roentgen picture. The suppleness of the intestine is preserved, the intestine may be distended, and the opaque column does not deform it. Again the appearance of the folds is of great diagnostic value. In the reactionary stage, even though there are changes in the number and the caliber of the folds, their regularity is preserved. In mucocatarrhal colitis the appearance resembles that of the irrito-exudative stage, but the folds are again loose and there is a suggestion of disorientation of the folds (Fig. 6). After insufflation there is a breaking up of the flocculant coating due to a retraction of of the gel on the collections of mucus (Fig. 7). The mucocatarrhal colitis may be more or less intense and in proportion to the stage of the lesion there is disorientation of the folds. Finally on the disorientation of the folds these follows a stage of irregularity of the folds, the transitional stage between mucocatarrhal colitis and ulcerative colitis.

NONSPECIFIC ULCERATIVE COLITIS

In ulcerative colitis it is necessary to distinguish different degrees, the period of onset, the period in which the lesion is fully

FIG. 6. Mucocatarrhal colitis; the coils are loose and widened, tending to lose their regularity.

developed, and the period of organization.

Period of Onset. The mucosa is destroyed, the folds have disappeared and there is a false folding; this is due to an unequal coating of the surface of the intestine by the opaque medium (Fig. 8). Insufflation is indispensable, because it allows demonstration of the inequality of the expansibility of the intestinal wall.

Period in Which the Lesion is Fully Developed. The ulcerations are more extensive and deeper and have a tendency to confluence. In the film taken at refilling the borders are irregular, the walls are stiff, jagged, and have spicules, and the whole picture may be that of double contour of the colon (Dell'Acqua).

In the collapsed condition there is found false folding, but to a more pronounced degree than in the preceding stage. The shadows follow no system.

Fig. 7. The same patient after insufflation; multiple spots due to conglomeration of mucus, striping of the borders discontinuous.

Fig. 8. A patient with left-sided colitis and blood in the stools. In the sigmoid disappearance of the folds with false folding; early ulcerative colitis.

Period of Organization. The walls react to the inflammatory process, and this defense reaction results in thickening of the walls and in polypoid proliferation of the intestinal surface. There result then the sclerohyperplastic lesions.

On refilling one finds that the intestinal caliber is decreased, the borders are fuzzy, notched, and bristling with spicules. Collapse is often imperfect because of the decreased suppleness of the walls.

On insufflation the decreased suppleness of the walls again calls attention to the polypoid appearance of the surface of the intestine which is more outstanding than in collapse.

TUBERCULOSIS OF THE COLON

Since the first description by Stierlin in 1910 of the findings obtained after ingestion much progress has been made.

From the study of the mucosa we are able in cases of tuberculosis to determine precisely the site of the lesions, their extent and their appearance. Given a patient

Fig. 9. Tuberculosis of the colon; persistence of the folds as far as the hepatic angle, then a segmented transition and finally false folding, ulcerated zone. Disappearance of the folds in the terminal ileum.

suspected of having intestinal tuberculosis, it is necessary to make a rectal exploration, which should always be followed by an examination from above to determine the condition of the small intestine.

The essential processes in tuberculosis of the colon are ulceration, plastic reactions, and exudation. According to the predominance of one of these three processes, variable findings will be obtained, but one must be on the lookout for transitional stages.

(1) *Ulcero-exudative Form, and the Ulcero-hyperplastic and Exudative Form.* I shall discuss together the ulcero-exudative and the ulcero-hyperplastic and exudative forms because pure types exist very rarely. There is in some cases predominance of ulcero-hyperplastic lesions. Among the patients we have examined it seems to be exceptional not to find involvement of the large intestine. In about 40 cases of tuberculosis of the colon which involved the right colon we have found lesions in the sigmoid in only two. This is due to the fact that Peyer's patches are the site of predilection for bacillary lesions.

In the description of the lesions I shall discuss only the most characteristic findings.

The Base of the Cecum: There is a great similarity between the various roentgenograms we have obtained. The base of the cecum instead of being rounded has a funnel-shaped deformity (Figs. 9 and 12). The deformity varies according to the

Fig. 10. The same patient after insufflation. All the segments are distended, except the base of the cecum which has a funnel-shaped deformity.

Fig. 11. The same enlarged. Good demonstration of ulcero-hyperplastic lesions. The valve of Bauhin is visible. Parietal reactions in the terminal ileum.

severity of the ulcerations and the reaction of the walls of the intestine.

When the lesions are predominantly ulcerative, the diameter of the intestine after filling is about normal, and the borders are finely jagged. In the collapsed state there is found in the ulcerated area a disappearance of the folds and a false folding, and after insufflation, the border of the colon, instead of being a dark continuous shadow, is puffy, discontinuous, and cut up by small gaps (Fig. 10). Finally, the intermarginal portion, instead of having a homogeneous shadow, has an irregular spotted or marbled appearance (Figs. 11 and 13).

If the hyperplastic lesions are farther advanced, the suppleness of the colon is decreased, and the intestinal lumen is narrowed. But that which demonstrates the rigidity of the wall is the fact that after filling, evacuation, and insufflation, the air in the cecum is practically unchanged.

Fig. 12. Cecal tuberculosis. Disappearance of the folds as far as the hepatic angle. The base of the cecum has no folds and has a funnel-shaped deformity. Disappearance of the folds in the terminal ileum.

Fig. 13. Same patient after insufflation. Sudden disappearance of the mucosal striping, the edges are irregular, the base of the cecum has irregular spotting. Still shows striping.

Lesions Adjacent to the Base: The base of the cecum, as I have already said, is the segment which is most frequently involved, but it is rare that the lesions are confined to it alone. Almost always they extend along the ileocecal portion and may extend to some degree even onto the transverse colon. The roentgenographic appearance may be the same as it is in the base, or it may be different, and sometimes, and this is the rule in majority of cases, it has a transitional appearance.

This transitional appearance may begin at the base of the cecum, the intestinal diameter progressively takes on normal dimensions, and the appearance of the mucosa passes through all the intermediate stages between the pathologic and the normal, or, strictly speaking, the subnormal.

Distal Lesions: The entire colon should be examined segment by segment, for any case of intestinal tuberculosis may show up at a distant point and in one which seems grossly normal. These secondary lesions which we have considered in the discussion of the reactionary conditions have nothing about them that is specific. The mucosa reacts by taking on an irritative or exudative appearance, or what is the most frequent, an irrito-exudative appearance, of which we have already given a brief description.

Terminal Ileum: When the large intestine is involved there are almost always concomitant lesions in the small intestine also (Figs. 11 and 12). And since by rectal examination we obtain in about 80 per cent of the cases a reflux into the terminal ileum it is possible to determine the condition of this segment. The lesions are of the same types, ulcerative and hyperplastic processes. Besides the usual types which I have just described, tuberculosis may also present other appearances, some of which I shall describe.

(2) *The Hyperplastic and Fibrous Form.* This is a very rare type. It is not always secondary to pulmonary tuberculosis and we think it is more often primary than is commonly believed. The lesions are localized in a short segment of the large intestine, commonly in the cecum, they are accompanied by well localized reactionary processes, and they have the roentgenologic characteristics of tumors.

(3) *Intermediate Forms.* I have emphasized the fact that tuberculosis produces polymorphous lesions and numerous transitional forms. The intermediate forms have a roentgenologic appearance which is a combination of various signs. However, among them there are two which are so distinctive that I cannot pass them by without mention. The first is the micro-colic form. As the term indicates, there is a diminution of the length and the caliber of the intestine, a condition which is found also in amebic colitis. The other is the ulcero-polypoid form which resembles very closely ulcerative colitis at the stage of organization. Often it is by clinical data alone that a differential diagnosis can be made.

CANCER OF THE COLON

The ordinary technique with the barium enema affords daily proof of its value in the diagnosis of cancer. But there are some cases where the method fails, yet cancer is present (Fig. 14). This is true in those cases where the tumor is small or where the opaque liquid obscures the small neoplastic outgrowths. In the diagnosis of cancer the study of the folds serves two important purposes: first, it may make an early diagnosis possible, and second it may outline the limits of the lesion, determine its operability, and in some cases furnish the indications for the type of operation.

From a roentgenologic point of view, cancer of the colon may be divided into four groups. This classification has the merit of differentiating the various roentgen findings and is not based on their histology. The main purpose of the roentgen study is to determine the various gross appearances of lesions and therefore allows differentiation on a basis of the most characteristic appearances, for example, a scirrhous from a nodular lesion.

I shall omit a discussion of the common findings and confine myself to a presentation of some new data which are brought out by a study of the mucosa.

(1) *Cauliflower Cancer.* This is the most common form and occurs in about two-thirds of the cases. In this group the value of the method of examination is well brought out, for it enables one to definitely state the limits of the lesion, which is of greatest value in the second and third stages of the examination.

After evacuation, it is common to see segments which appeared normal at the first stages of the examination show definite signs of malignant infiltration.

In the cauliflower growth there are no folds, for the epithelium of the mucosa is destroyed, but the roentgenographic image

is made up of spots, more or less puffy, which present an alveolar appearance. There are spots which may be clear, oval or rounded, surrounded by a dense dark striping. When the lumen of the colon is reduced the pattern becomes indistinct, the various structures cross into each other and all systemization is lost, but some clear spots, usually rounded, can be distinguished with difficulty.

In the study of cancer the use of the method of insufflation is almost indispensable, and is the method of choice for determining the extent of the infiltration, for all the segments which are infiltrated are rigid and cannot be completely distended. Moreover, in those areas where there is only an infiltration of the submucosa the striping of the mucosa is preserved, whereas in those cases where the mucosa is destroyed the surface is nodular, there is no longer any striping of the mucosa or else it is very irregular.

(2) *Mulberry Cancer.* This is a little rounded, sessile, multilobed tumor. The mucosa is destroyed over a small area. This is the initial stage of cauliflower cancer.

On filling, the cancer can be seen only when it is on one of the margins, and one can visualize not the tumor, but its point of implantation into the bowel. For that reason it may be missed.

In the collapsed stage, its appearance is that of a mulberry, with a finely granular surface. But it is only after insufflation that one can see the extent of the tumor, for then it stands out definitely from the shadow of the bowel.

(3) *Papillary Cancer.* Here the diagnosis is easy, for the stenosis is serrated and no matter what the method of examination it will usually permit definite diagnosis. But the great advantage of flocculent solutions is that their fluidity causes them to penetrate into the serrated stenoses even when there is a substricture of the segment. Therefore the limits of this tumor can be marked out exactly. On insufflation the liquid carried in with the air accentuates the contrasts greatly, and shows conclusively that the tumor is rigid.

(4) *Infiltrating or Sheath Cancer.* This is the rarest form of cancer of the colon. The neoplastic infiltration involves a long segment of the colon, sometimes for a distance

Fig. 14. Cancer of the cecum, verified by operation. The tumor is represented by a circular shadow in which light spots can be seen, cauliflower growth. Tumor invisible on filling.

of 12 to 15 cm. Even though there is little change in the caliber of the bowel for a long time the evolution of the lesion is slowly toward a stenosis.

In the early stages the tumor develops in the submucosa, and destroys the mucosa in isolated areas, but the wall is rigid. On filling, since the lumen of the bowel is normal, the tumor is often missed. In the collapsed stage, the folds persist, but the folding is poor as a result of infiltration of the submucosa, and moreover it is sometimes possible to see small opaque or clear spots over scattered areas due to crypts or buds. But here again it is insufflation which

permits definite diagnosis by outlining the rigid segment and the irregularity of the shadow of the bowel.

BENIGN TUMORS—POLYPS

Benign tumors of the colon are relatively rare and they are more of anatomic than of roentgenographic importance. Their

Fig. 15. Roentgenogram after evacuation. Alveolar appearance of the whole left colon due to a micropolyposis, proved by rectoscopy.

rarity is probably due to the fact that ordinary methods of examination miss many cases of solitary polyps and of polyposis.

(1) *Non-confluent Benign Tumors or Polyps*. Most of the roentgenologic studies of polyps are of recent date (Charrier and Calderon, Györgyi, Hellmer, Knop, Pansdorf, and others). All authors who have succeeded in demonstrating polyps have used a method very similar to ours. In fact, a polyp the size of which may vary from that of a grain of wheat to that of a nut is often hidden by the excess of opaque liquid in the distended intestine. Polyps are sessile or pedunculated, some are solitary, others are double, triple or quadruple. The site of predilection is the sigmoid. On filling, if their size is sufficient they may stand out as bright spots from the rest of the opaque field. But it is especially at collapse and after insufflation that they are discovered. In the stage of collapse the intestinal wall covers the polyp which is then represented by a rounded or oval spot surrounded by a dark zone. The folds take on a circular arrangement. On insufflation the reverse takes place, the polyp then standing out in gray from the clear background. If the pedicle is long the polyp may be displaced in the distended bowel. The surface of a benign tumor is always smooth, a finding different from that in malignant tumors or in coproliths.

(2) *Polyposis or Benign Confluent Tumors*. Polyposis is more common than solitary polyps. It is familial and is generally diagnosed between the ages of ten and forty. Bensaude's school has reported the most complete description of this lesion. The affection may be localized in one segment of the bowel or it may extend throughout its length, or it may involve isolated segments with normal bowel between them.

On filling, the less force there is used, the better the chance of visualizing the polyp. The largest of them present the appearance of a rounded clear spot like that of isolated polyps. In cases of polyposis full filling of the bowel is of little value, and Bensaude in 1931 pointed out the advisability of omitting the enema study and of relying on insufflation of the colon.

In the collapsed state, the picture is markedly changed. In place of the normal folding there is a typical reticular appearance. The links touch each other. The interior is clear, the strands are sharp, and of the same size. The links are not all of the same diameter, but they are usually single or double. The network is homo-

geneous, and composed of rounded or polygonal segments. It is common to have folds of an irrito-exudative type (Figs. 15 and 16) in the intact segments.

After insufflation, according to the severity of the process, the suppleness of the walls and their expansibility are more or less preserved. Throughout the whole involved segment there is a crop of opaque rings surrounded by a clear background. The rings may be contiguous or tangent.

DIVERTICULOSIS AND DIVERTICULITIS

According to their etiology diverticula are classified into three groups: (1) traction diverticula; (2) pulsion diverticula; (3) congenital diverticula.

Traction Diverticula. These should be called false diverticula for just as in the esophagus these diverticula are the result of external traction. They do not meet the true definition of a diverticulum, that is, a hernia of the mucosa and submucosa through the muscularis. They are almost invisible after the opaque enema or after gaseous distention, but are visualized after collapse, a stage in which the loops are opposed to their retractor. They are usually finger or cone shaped. The mucosal folds converge toward the summit of the shadow to which point it is sometimes possible to follow them.

Pulsion and Congenital Diverticula. These two groups can be studied together for their roentgen appearance is the same, and their differentiation is impossible. To this group belongs properly the term diverticulosis, or, if there is an inflammation present in them, diverticulitis. In this syndrome also the study of the folds is the method of choice, for it is the only method by which the lesions can be definitely detected, inflammatory processes in them can be appreciated, and a decision permitted as to whether this process is primary or secondary.

Diverticulosis. The diagnosis of diverticulosis is simple in that it lies almost exclusively in the demonstration of diverticula. The more the intestine is distended, the less the chance of recognizing the diverticula. For those on the face of the bowel are masked by the opaque column, and only those which are tangent to it can be seen.

After evacuation, the collapsed intestine is reduced to its smallest surface area and

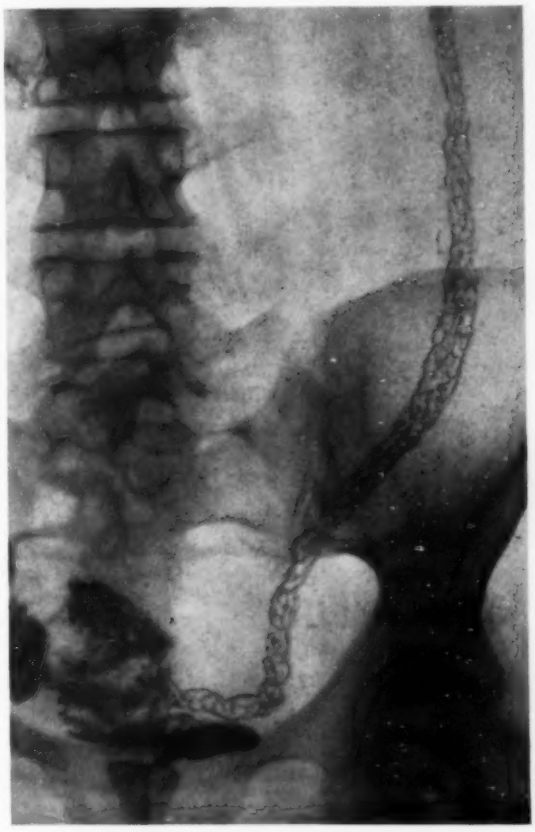

Fig. 16. The same patient after evacuation of a barium sulphate enema. No sharpness of the shadows, diagnosis of polyposis almost impossible.

all the diverticula whose necks are permeable are visible. In certain cases one may see the mucosal folds converge toward the point of implantation of the diverticulum, but they do not enter the diverticulum proper, as is sometimes the case with traction diverticula. After insufflation if they have kept their opaque contents they are distinctly outlined. The diverticula in the flat surfaces of the colon stand out in black against the clear colon distended by gas. In other cases, the air may be substi-

tuted for the opaque medium in the cavity of the diverticulum, in which case they appear as a clear bubble surrounded by a dark area. But the cavity of the diverticulum is not always empty, for stercoliths, or particles of organic matter may occupy all or part of the cavity, when the picture

FIG. 17. Patient with diverticulosis; multiple filled diverticula. The colon is supple and well distended. Striping of the mucosa normal.

is different, for the quantity of liquid which may get in around stercoliths varies from one diverticulum to another.

In order to make a diagnosis of diverticulosis, it is necessary not only to study the diverticula but also the mucosa near them and at some distance from them. This is the most important part of the examination, for through it we find the elements that permit making a differential diagnosis of diverticulosis from diverticulitis. In diverticulosis axial symmetry is preserved, the caliber and the number of the folds are within normal limits. After insufflation the distention is good, and the mucosal striping is preserved (Fig. 17).

Diverticulitis. The clinical findings alone may often raise a suspicion of this lesion. Diverticulosis is painless whereas diverticulitis is painful.

The diverticula have the same appearance as in diverticulosis, except in those advanced cases where the diverticulum has been transformed into an abscess.

The appearance of the mucosa, depending on the degree of inflammation, may be that found in the irrito-exudative stage or that in ulcerative colitis. The folds tend toward disorientation, to complete disarrangement and to false folding. Inflamed diverticula may be surrounded by a more or less rigid ring comparable to a neoplastic infiltration. It is impossible to get a collapse of the bowel, and this increases the difficulties of diagnosis. This condition is most often seen in the sigmoid, and is the most common cause of stenosing sigmoiditis.

CONCLUSIONS

The interpretation of some of the roentgenologic appearances of the mucosa of the colon has been attempted. It has been my intention to point out that some of these findings are not as complicated as they may seem but are easily referable to their correct anatomic or physiologic basis. The technique of examination is a delicate one, and one which unfortunately is not applicable to all cases. It is, however, indispensable because of the richness of the detail which it brings out.

The technique of the study of the folds has not yet reached its greatest development and it is only by greater experience that we shall be able to reach a correct interpretation of the evidence which it furnishes.*

* All the roentgenograms were furnished by the Hopital Laennec, Paris. Medicin-chef, Dr. G. Maingot.

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DIRECTED ROENTGENOGRAPHY OF THE THORAX*

(THE CARDIOCAIROGRAPH)

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BY A DIRECTED chest exposure is meant an exposure of the chest at a predetermined phase of the cardiac cycle. This is best accomplished by the synchronization of the exposure with a selected phase of the circulatory phenomena, heart sounds, pulse waves or action current impulse. The efforts to accomplish this are by no means recent for as early as 1925 attempts were made to expose the chest at a certain definite instant either by utilizing the carotid pulse (Weyl, Warren and O'Neill[8]) or the heart sounds (Rivolta). These methods suffer from an inherent difficulty. Both the heart sounds and the pulse waves occur during systole, and in order to synchronize with the end phase of diastole, the desirable instant, it is necessary to introduce a delay mechanism, the magnitude of which depends upon the pulse rate. This, even in the same individual, may be variable so that no uniform results can be expected by these methods as the adjustment for pulse rate cannot be accurate or exact.

Further, the difficulty of timing by means of heart sounds lies in the interference produced by extraneous noises, as well as in the variable quality of the first and second sound.

The action current has been used as a control by timing the disturbance produced by the roentgen exposure on simultaneously recorded electrocardiograms. Bergk and Chantraine[1] used action currents to set off the exposure. Their apparatus will be referred to below.

The method here described also makes use of the action currents of the heart (Hirsch and Schwarzschild[4]). The R wave is utilized to set off the circuit closing mechanism which makes the roentgen exposure.

The method is simple, positive in action and easy of application. The device constructed to accomplish the results is called the cardiocairograph (from the Greek "kairos" meaning a definite point in time, c being substituted for k in order to avoid confusion of the word "cairograph" with "kymograph").

The action currents are taken from the patient through the standard leads of the electrocardiograph. Correlation of the roentgen kymographic movement wave with the electrocardiographic phenomena[3] has shown that the peak of the R wave is attained about 0.08 second before the beginning of the systolic motion. The peak of the R wave may therefore be considered to correspond to the time of maximum diastole. It is necessary to define the meaning of the terms systole and diastole, as usually used, and their connotation in connection with this work. Ordinarily systole indicates the time of tension of the cardiac musculature, while diastole commonly implies the period of relaxation of this musculature. But for our purposes systole must be taken to signify the interval during which the ventricular musculature is contracting, and diastole that part of the cardiac cycle during which the musculature of the ventricle is expanding.

In order to obtain a roentgenogram of the thorax during the time when the heart is in maximum diastole it is thus only necessary to arrange for the exposure to take place at the peak of the R wave, or shortly thereafter. The apparatus herein described is adapted for this purpose and thus permits directed exposures at maximum diastole without any time-delay such as is necessary if the carotid or other pulse waves or heart sounds are utilized for closing the roentgen-ray circuit.

* From the Department of Radiology, Beth Israel Hospital, New York.

However, to produce directed exposures at maximum systole a time-delay mechanism is used. The time-delay required between the peak of the R wave and the time of maximum systole is relatively constant, being about 0.20 second and essentially independent of the pulse rate within wide limits. The reason for this lies in the fact that this interval is closely related to the conduction excitation time of the cardiac muscle and does not extend over the isoelectric or diastolic interval, which is the part of the cycle that varies most markedly with the pulse rate. Thus, diastole or systole may be utilized in the same cardiac cycle, and the errors which might arise from the variation in the pulse rate are reduced.

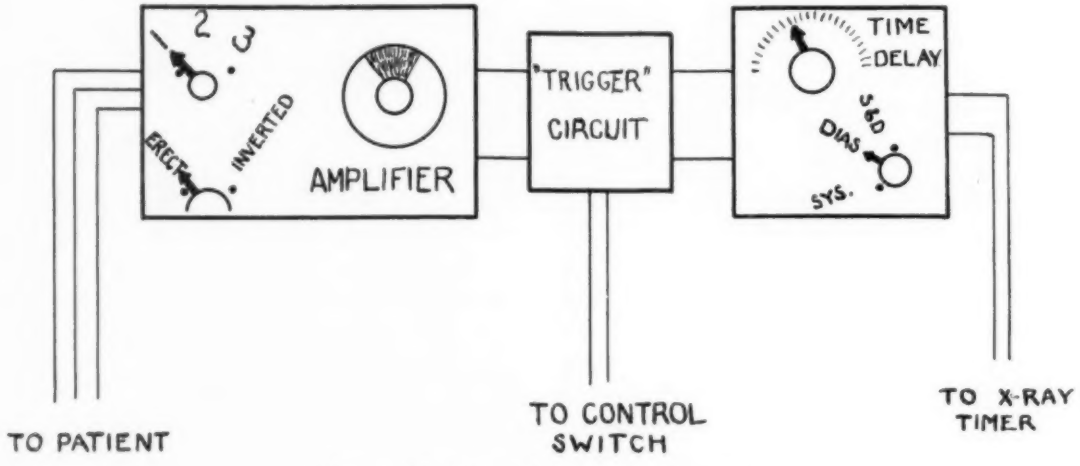


FIG. 1. The plan of the cardiocairograph.

Figure 1 illustrates the plan of the apparatus. The patient's leads are connected to an amplifier which is equipped with a special cathode-ray tube.* The indicator of the tube is in the form of a fluorescent lighted sector covering the whole area of the target, and is viewed through the top of the bulb. During the operation of the tube the two edges of the lighted portion separate with a fan-like motion leaving an unilluminated sector on the conical plate.† This tube enables the operator to watch the action currents and select intelligently the most suitable lead to be used. That lead is utilized which has the largest R deflection. This is easily determined. Should there be an inversion of the deflection in the selected lead it is shown in the cathode-ray tube, and the leads are accordingly interchanged by simply throwing a switch arranged for that purpose.

The output of the amplifier, which is some ten thousand times greater than the input voltage, is fed into a "trigger" circuit. The essential elements of the trigger circuit are a thyratron tube and a condenser. The thyratron tube is a gas-filled triode which has the property that the plate-to-cathode path is open as long as the grid potential is maintained below a certain fixed level, whereas if the grid, even momentarily, is raised in potential above this fixed level, the plate-to-cathode path becomes conducting and remains so until the external plate circuit is interrupted. Figure 2 illustrates the course of events about to be described. With the patient connected and the amplifier operating, the magnified electrocardiographic waves are impressed upon the grid of the thyratron in such a manner that the peaks never reach the flashover potential referred to above. When the control switch is closed two things occur. First, the plate circuit of the thyratron is completed, and second,

* The "Magic Eye," a cathode-ray type of thermionic vacuum tube (Type 6E5).

† This tube is used in many radio sets as a visual tuning indicator.

a condenser, which up to this time has been charged and depressing the grid potential, begins to discharge. The grid potential thus rises, as indicated in the figure, and the thyratron tube flashes over at the instant the grid attains the flashover potential. A consideration of the figure shows that this obviously must occur near the peak of the ascending limb of an R wave.

The use of this "trigger" circuit, with no inherent time delays, is the heart of our proposal and distinguishes it strongly from the arrangement of Bergk and Chantraine. Their apparatus includes relays with inherent time lags, so great as to necessitate the use of time delay, covering practically a complete cycle for the diastolic exposure.

The flashover phenomenon in the thyratron tube permits current to flow in the plate circuit of that tube. This circuit includes a time-delay mechanism and the roentgen-ray timer. The time-delay mechanism is equipped with a three-point switch so arranged that in the systolic position a predetermined time-delay, usually 0.20 second, is introduced between the flashover and the closing of the roentgen-ray contactor. In the diastolic position the time-delay device is out of the circuit and the flashover of the thyratron tube causes the immediate activation of the roentgen-ray circuit. In the third position two roentgen exposures are made: one directly actuated by the flashover, and the other a predetermined time (0.20 seconds) thereafter.

It is thus possible, with the cardiocairograph, to make exposures, either in systole or in diastole, or if desired, to make two exposures on one film to show the variation in the cardiac shadow between the two phases.

In some cases the R wave does not extend above the T wave in any lead. While this is rare, it is sufficiently important to require a remedy. The time of the peak of the R wave being the only point of interest, the amplifier is so arranged that the rapid deflections of the R waves are amplified to a much greater degree than the slower waves of the electrocardiogram. In this way the potential variations reaching the thyratron grid are not a true magnification of the electrocardiogram but rather a distorted representation in which the important R waves are highly magnified while the undesirable P and T waves are relatively suppressed. The time of the R wave is, however, unchanged.

For satisfactory operation of this device

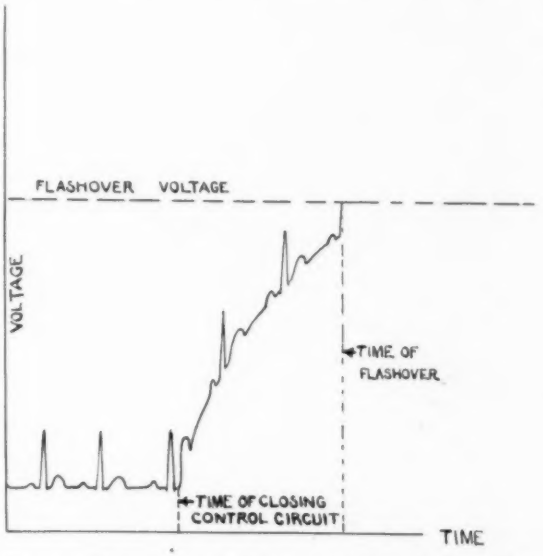


Fig. 2. The course of the potential variation of the grid of the thyratron during the period just preceding the roentgen exposure.

the roentgen-ray contactor and its associated mechanism must be extremely rapid in response. Ordinary timers are entirely unsatisfactory, because of the variations in time of starting of from one-thirtieth to one-quarter of a second. These variations arise from the necessary multiplicity of relays and moving parts in any available timer system adequate to handle the energy necessary for rapid chest exposures with accurate timing. The timer used for this work is based on principles discussed by one of us some years ago (Schwarzschild[7]). The roentgen-ray contactor of the timer is directed by a very light, rapid acting relay in the thyratron circuit. (If necessary this relay may also be dispensed with.) When the trigger circuit acts the roentgen-ray contactor is caused to close and the exposure begins.

The opening of the roentgen-ray contactor at the end of the exposure is brought about by the discharge of a glow-tube through another relay. The glow-tube is discharged when the roentgen tube has passed a predetermined number of milliampere-seconds. This is accomplished by allowing the roentgen tube current to charge a condenser. Further details of this device will be given in another communication. All the cardiocairograms were made with 8 ma-sec., 200 ma., at a distance of 4 feet, with a Rotalix tube.

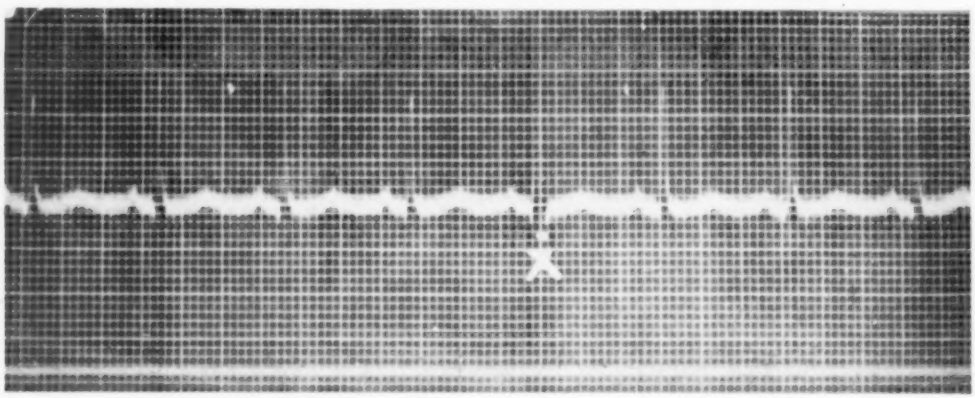


FIG. 3. An electrocardiogram taken on a patient simultaneously with a cardiocairogram. A break in the electrocardiographic tracing is noticed at the point *X* corresponding to an R wave, showing that the cardiocairograph actually takes the roentgen exposure at the time of the R wave when used without time-delay.

To judge the results obtained with this device it is necessary to prove (1) that the cardiocairograph accomplishes the mechanical result for which it was designed, and (2) that the resulting roentgenograms are in the proper phase of cardiac action.

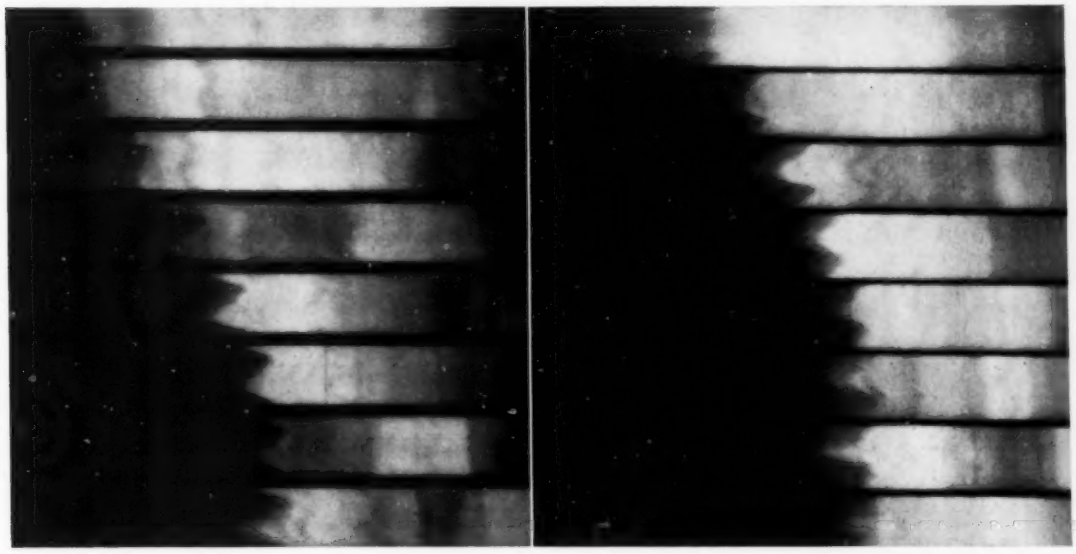
As regards the former consideration, Figure 3 shows an electrocardiogram taken with the string galvanometer on a patient who was simultaneously connected with the cardiocairograph. The point marked *X* indicates the time at which high tension was applied to the roentgen tube, since at that moment the string of the galvanometer was thrown off the field by the induced voltage from the roentgen tube leads. It is to be noted that the roentgen exposure takes place before the downstroke of the R wave. Thus we may say that the original mechanical requirements have been met.

This method of checking may be used for controlling the apparatus in order to determine after each exposure whether it was taken during a normal wave or during an extrasystole or other abnormal wave. A modification which does away with the necessity of photographic processing involves the use of a cathode-ray oscillograph with lag screen. With this device a temporary fluorescent image of the electrocardiogram is produced upon which the point of roentgen exposure is clearly indicated. For this purpose one pair (vertical) of deflecting plates is connected to the amplifier output, while the other is connected to a slow sweep circuit which sweeps the beam across the screen at a speed of about 1 inch per second.

Through the courtesy of Allan B. Du Mont we have been able to use his oscillograph tube and to demonstrate its value for this purpose.

In order to prove that the points of time selected are really those of diastole and systole, checks were made on a number of subjects, using the kymogram as a control. These were made by a technique previously described. The method consists essentially in moving a film in the vertical direction during the course of a one second exposure of the thorax, a lead sheet bearing a series

of narrow slits about 1 cm. apart being interposed between the film and the subject. During the one second exposure the film moves a distance slightly less than that between adjacent slits. The resulting film thus shows a series of tracings of the movement of the cardiac contour during the time of exposure. In each horizontal band of the kymogram the exposure starts at the bottom and the motion of the point of the contour is to be followed from the bottom upward. With the view to checking our device, the cardiocairograph was arranged to set off the kymographic exposure. In Figure 4*a* is shown a series of waves with the device set to start the exposure at maximum diastole. It will be noted that along the ventricular border the beginning of each band is actually at the point of maximum diastole. In Figure 4*b* matters were arranged to set off the kymograph at maximum systole. It will be noted that the records of the ventricular border actually are in the trough of the wave, or maximum systole.



a *b*

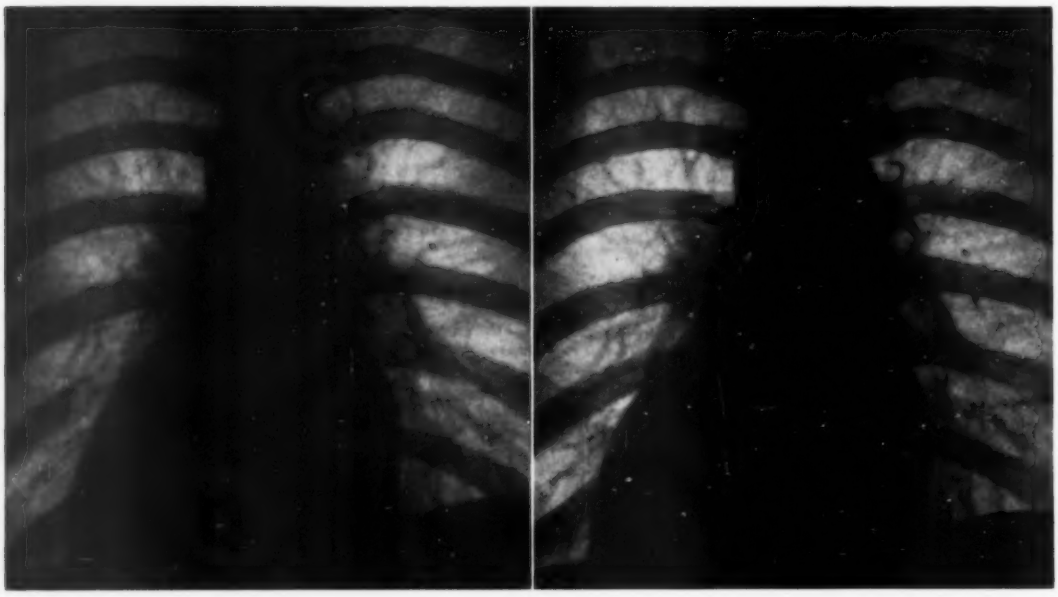
Fig. 4, *a* and *b*. Cairokymograms showing part of left ventricular border. In *a* the kymographic exposure was set off by the cardiocairograph set for diastole (no time-delay). Note that in each ventricular band the record starts in the maximum diastolic position of the ventricle. In *b*, the kymogram was set off by the cardiocairograph set for systole (with time-delay). Note that in each ventricular band the record starts in the trough of the wave, or maximum systole.

The method here described for making kymographic exposure at a definite point in the cardiac cycle as determined by the action current phenomenon may be of assistance in the study of the details of cardiac movement. In all such cairokymograms, as such films may be called, the wave motion begins at the same definite point in the cardiac cycle in the bands of different films.

In studying kymograms in the past it has always been necessary to correlate the beginning of systole with the beginning of the outthrust of the vascular wave, or with the sound notch.[5] In many kymograms these waves or notches are not well defined. In such cases the cairokymogram would be of great value.

Further, it provides a simple method for tying up the electrocardiograph with the kymograph free from any of the difficulties involved in the previous methods used by us,[3] since there is no shielding problem in this procedure.

Figures 5*a* and *b* show a pair of films taken one immediately after the other, without breathing, but in opposite phases



a *b*

FIG. 5, *a* and *b*. Cardiocairograms taken in opposite phases of cardiac action; *a* systole, *b* diastole. The change in the cardiac contour is apparent.

of cardiac action. A change in the cardiac contour is apparent. Figure 6 shows a double exposure, systole and diastole, in a case with considerable amplitude of cardiac motion. The difference in shape of the two contours is evident in spite of the absence of any change in the position of the diaphragm or ribs. The two exposures were made within 0.20 second of each other and represent the diastolic and systolic contours of the *same* cardiac cycle.

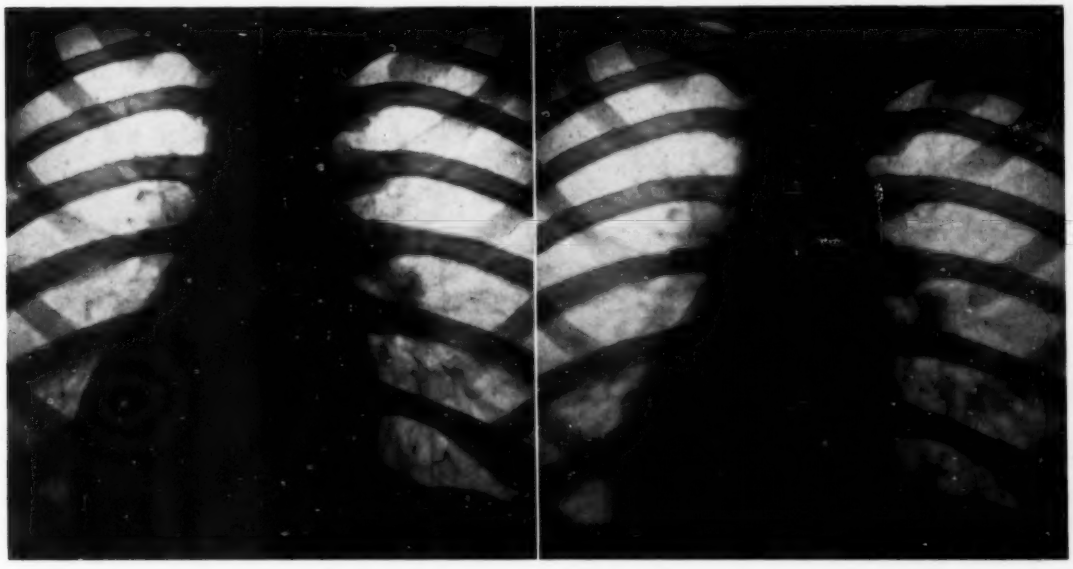
FIG. 6. A double exposure, systole and diastole, showing the differences in cardiac contour. The two exposures were made on one film during the same cardiac cycle.

This method permits us to correct an old fallacy which, however, still seems to be general. It is usually stated that a long exposure through several cardiac cycles would give what was considered to be a shadow of the heart in maximum diastole. The reasoning on which this conclusion was based is faulty since the border of the heart in long exposures will naturally be the border bounding the unexposed region. This border is obviously that of the heart when in maximum systole. Since, however, there is no sharp border at all in the long exposures one subjectively selects an apparent border which lies somewhere between systole and diastole. Usually this apparent border is quite different from the diastolic border. This is shown by Figures 7*a* and *b*. Figure 7*a* represents the heart of a young adult in diastole taken

with the cardiocairograph (1/30 second) while Figure 7*b* shows the same heart taken immediately after and with a one second exposure in the same respiratory phase. The contours are definitely different, the long exposure giving a heart contour which is not that of diastole.

For comparison of cardiac contour, from time to time, in the same individual, a standardized method for taking such film, always in diastole, is obviously desirable. There remains, however, a serious obstacle in that it is difficult if not impossible at present, to insure the making of the exposure in the same respiratory phase. The importance of this factor was demonstrated by making two films, both in diastole, within a few seconds of each other but in opposite phases of respiration. There was diaphragmatic movement, the thoracic cage being unchanged in position in the two films. The cardiac contours were not alike. For comparison a pair of films, both also in diastole, were taken about an hour apart. The cardiac contours were exactly alike. The subject was requested to duplicate the respiratory phase. This procedure is feasible with intelligent subjects, but for ordinary cases conditions are best duplicated at deepest inspiration. This has been verified repeatedly.

It has long been recognized in chest roentgenology that the motion of and within a patient is the most important single factor in the control of definition and sharpness of the image. The amount of blurring and unsharpness produced by movement varies with the rate of movement and with the duration of the exposure. If the movement of the patient cannot be controlled the remedy applied is shortening of the exposure. But this does not take care of such movements as rise from the impact of the pulsating heart against the lungs and the pulsation of the vessels. This motion varies in relation to the cycle



a *b*

Fig. 7, *a* and *b*. *a*. A cardiocairogram in diastole and *b* simple film of the same chest taken immediately after *a* with an exposure of one second. Note the difference in the size of the cardiac contour. It is apparent that the long exposure procedure does not give a shadow of the heart in diastole. These films demonstrate the improvement in definition and detail due to the short exposure. All the unsharpness in *b* is due to vascular movement (Bouwers, Wilsey). The kilovoltage and distance in the two exposures were exactly the same. The time and milliamperage were naturally varied. That the subject did not move is evident from the sharpness of detail in ribs and diaphragm in *b*.

of the heart beat and varies in different portions of the lungs, depending on the proximity of the vessels to the heart. While at the periphery in certain parts of the lung, the movement may be minimal, it may be as rapid as 20 mm. per second at the apex. Besides this, the extent of the movement varies in different individuals, and in the same individual from time to time.

A study of the pulsatory movements readily discloses the fact that the movement of the lung structure is at a minimum at that instant when the ventricles are almost at the end of the diastolic phase. This therefore is the ideal point of time for the roentgen exposure of the lung. Such directed exposures, in contrast to the fortuitous or undirected exposures as now made, are thus eminently desirable if it is the purpose to obtain the maximum degree of sharpness of detail of the pulmonic structures.

McPhedran and Weyl,[6] Bouwers,[2] Bergk and Chantraine,[1] and Wilsey,[9] have discussed the extent of improvement of the detail of the lung roentgenogram to be expected from synchronization of the exposure with cardiac diastole. A large number of test exposures in diastole and systole have demonstrated the definite advantage of the diastolic exposure over the systolic. The lung markings are sharper and clearer and terminal markings may become visible in the diastolic exposure which cannot be seen in the systolic or the fortuitous exposure. The periphery of the fields becomes rich in detail. The method, therefore, has a practical application in the roentgenography of the lungs.

SUMMARY

An apparatus (the cardiocairograph) is described which synchronizes the roentgen exposures of the thorax with the cardiac action current phenomenon. This makes it possible to obtain roentgenograms in maximum diastole without the use of any time-delay, and to obtain roentgenograms in maximum systole with a time-delay covering only the systolic interval, reasonably constant from individual to individual and relatively independent of the pulse rate.

Another application of the cardiocairograph for the production of cairokymograms, i.e. kymograms, in which the time scales begin at any predetermined phase of the cardiac cycle, is described.

The variations in cardiac contour in their dependence upon cardiac and respiratory phase are described. It is pointed out that roentgenograms made in systole may show contours markedly different from those made in diastole. It is also shown that the phase of respiration has an appreciable effect upon the contours of the cardiac shadow. The use of the cardiocairograph during maximum inspiration provides a standard method for studies of the cardiac contour.

The method described also provides a means for checking the contours found by the long exposure procedure. Such exposures yield contours very different from that of diastole, approximating in many cases that of systole.

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CONGENITAL OR INFANTILE DISLOCATION OF THE HIPS*

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CONGENITAL dislocation of the hip, so-called, is a universally accepted entity, well established in the literature and known since the time of Hippocrates. It is one of the important conditions with which the orthopedic surgeon has to deal and quoting from the text by Jones and Lovett,[4] "The favorable prognosis that can now be given in suitable cases is one of the striking achievements of modern surgery."

Good to excellent results can be shown in a considerable percentage of cases by the advocates of either the open or closed methods of reduction, but it is also evident from late surveys that there is still a great deal to be hoped for.

Orthopedic opinion now seems to be quite unanimous with that expressed by Putti,[7] Freiberg,[4] and others, that the age of the patient when treatment is started is the most important factor in results. Putti would reduce, indeed abolish, the age limit completely, and begin treatment the very moment the deformity is observed even if that were the day of birth.

Of course before treatment can be started a diagnosis has to be established and upon our future knowledge of the condition much will depend on roentgenological studies starting at birth.

Numerous theories have been evolved regarding its etiology including trauma at birth, trauma before birth, primary nerve lesions and primary developmental defects.

Putti[6] thinks the dislocation is usually the result of chronic trauma to the lower extremities and hips during the later half of prenatal life.

Chapple[1] says the dislocation is not present at birth, but that the upper portion of the acetabulum is defectively developed and dislocation takes place only after force is applied.

My own conviction is that the deformity is usually a post-natal development due to an inherent relaxation of tendons and ligaments. It is predominant in females with a hereditary and familial tendency. All changes in bone and joint contours are secondary and cannot be demonstrated at birth. In other words, soft tissue relaxation allows a dislocation in an otherwise normal joint (Fig. 1).

Its development is comparable to that of the so-called flat, pronated, or weak feet so common in infants.

In both conditions there is a potential but not actual deformity before birth and in both there is a hereditary and familial tendency. Not infrequently, they occur in the same patient and may be associated with general hypotonia. One occurs at the upper end, the other at the lower end of an organ of locomotion and weight bearing.

In order to appreciate the mechanics concerned in the development of this deformity I will consider briefly certain features of the anatomy of the hip.

It is the truest ball and socket joint in the body. Proper position of the articulating surfaces is very much dependent on the relative size and shape of the opposing surfaces. Motion is restricted to well-defined limits and normally the joint is very stable.

At birth the whole upper end of the femur, head, neck and trochanters and a large part of the acetabulum are cartilaginous (Fig. 2). Growth is rapid and molding of these relatively soft structures is possible in a short space of time. Normal development is dependent on an accurately approximated and firmly applied femoral head. We see this beautifully demonstrated when an infant's dislocated hip is properly reduced (Fig. 3). Nature's response to normal stresses as well as to lack

* Read at the Thirty-seventh Annual Meeting, American Roentgen Ray Society, Cleveland, Ohio, Sept. 29–Oct. 2, 1936.

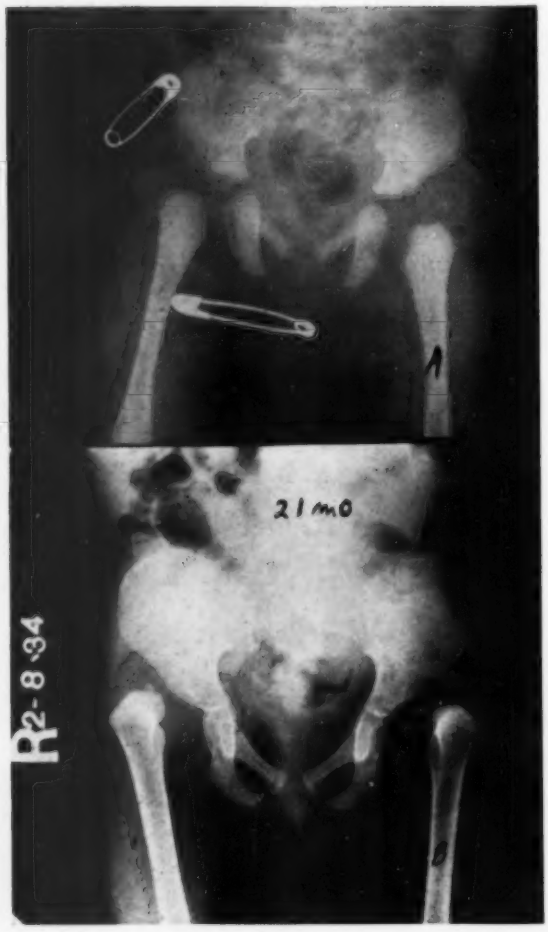

FIG. 1. *A*. Hips of a baby at the age of one week. The physician who delivered the case said labor was easy. Soon after birth he noticed that the hips were lax and could be dislocated and reduced easily. General tone of baby was poor. This film taken at another hospital was reported as being negative. Note that the acetabula are normal but there is distinct relaxation of the femoral heads. At eight weeks the baby was referred to the Children's Hospital and although the orthopedic resident had the previous physician's report he was unable to detect anything abnormal in the hips at that time. He did not even ask for a roentgenogram. *B* shows the hips when the child began to walk, completely dislocated. (I wish to thank Dr. Richard Collins of Waltham, Mass., for the original roentgenogram shown in *A*.)

of them is a striking feature at this age.

Now if, as the result of relaxation of soft tissues during the formative neonatal period, the joint surfaces are not held in proper apposition, the acetabulum, lacking the necessary stimulation from pressure of the femoral head, becomes relatively smaller and soon the ball does not fit the socket (Fig. 4).

As the child grows, muscular activity increases and tone of tendons and ligaments improves. However, the predominating muscle pull is not in direct line with the opposing joint surfaces so that instead of a natural tendency to spontaneous correction, as in most other joints, distortion results, disproportion increases and we have what amounts to a vicious cycle. Sometimes the forces are so counterbalanced that only partial dislocation results in a so-called congenital subluxation (Fig. 5). In other instances, the corrective forces predominate after a time and a subluxa-

Fig. 2. Normal hips before and after death at three days, dissected to show proportions of cartilage and bone. Most of the acetabular socket as well as the upper end of the femur, trochanters, neck and head are cartilaginous. The acetabulum accommodates about one-third of the femoral head at this period.

tion is spontaneously reduced (Fig. 6). Slomann,[8] Ducroquet,[3] and others, have reported a number of such cases. In roentgenograms of children's hips we run across some very shallow acetabula even though they are not dislocated. This shallowness I believe indicates a previous tendency to dislocate, but due to corrective forces dislocation was prevented. It has been suggested that the predominance of dislocations in females is due to a difference in the width of the pelves in the sexes. If there is any difference at birth it must be very slight, and it is very doubtful if this could account for the sex ratio. In association with extrophy of the bladder the symphysis pubis is always separated so that

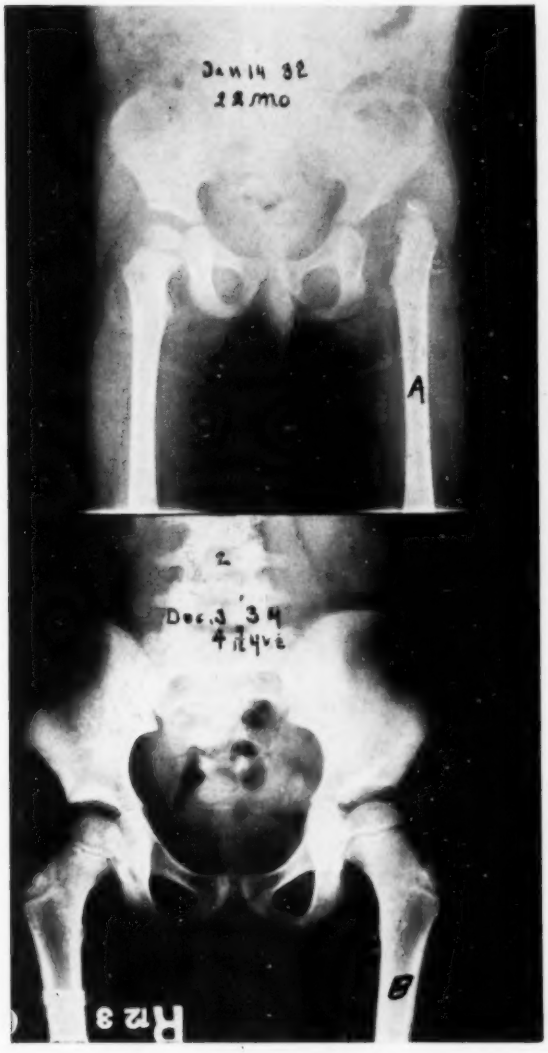

Fig. 3. *A*. Hips of a female infant at the age of twenty-two months who showed the typical clinical and roentgenological findings of "congenital dislocated left hip." *B*. The same patient two and a half years later following closed reduction, at which time the hip was normal, functionally as well as anatomically. This would seem to indicate that there had been no primary abnormality of the joint itself.

Fig. 4. *A*. Hips of a male infant with lumbar meningocele at the age of twelve days. There was no detectable paralysis of the extremities or sphincters. Note normal development of the acetabula and relaxation of the left femoral head. This feature was not recognized clinically. There was no apparent abnormal mobility even after the roentgenograms were seen. *B* shows the same case at one week less than four months. The meningocele had been removed, with excellent results. Note the marked difference in the acetabula and the dislocated hip on the left. This change has occurred in three and a half months.

FIG. 5. *A.* Female infant, aged fifteen months, apparently normal until beginning to walk at fourteen months when a painless limp was noticed on the right. Birth was called normal. There was piston mobility on the right side and positive Trendelenburg sign. Left side seemed normal to physical examination. Roentgenogram shows complete dislocation on the right and slight subluxation on the left with shallow acetabulum. Treatment was by the closed method with both hips in abduction. *B.* At four years function was normal.

the pelvis is very wide (Fig. 10). Of 40 such cases seen at the Children's Hospital during the last eighteen years, none had dislocated hips.

I have referred to the relaxation which I consider to be the usual initiating cause of the dislocation as being inherent. I see no reason, however, why the relaxation may not in some cases be due to trauma or possibly some other cause. In reviewing the records of some 259 cases of so-called congenital dislocation of the hip a rather large number had difficult birth histories. Of this group 17 per cent were breech deliveries and 13 per cent were instrumental.

We know that in the examination of a new-born baby significant injury may be

FIG. 6. *A.* Hips of a baby girl at the age of three months who was brought to the clinic on account of pronated feet. Note the shallowness of the acetabula and slight upward displacement of the femoral heads. General muscle tone was poor and there was marked diastasis recti. *B* shows the hips at fourteen months. Treatment consisted in the wearing of foot pads. Nothing was done to the hips.

overlooked, and also that a history obtained from the mother months or years later is apt to be vague about the neonatal period. Nevertheless birth trauma seems to be an important factor in certain cases (Fig. 7).

We have had a number of dislocated hips in patients with generalized hypotonia such as is associated with cerebral deficiency and cretinism (Figs. 8 and 9). The roentgenological findings are the same in all cases.

Fig. 7. *A.* Hips of a female infant at the age of three months. This had been a very difficult breech delivery. Soon after birth fullness was noted in the left hip region and there was pain when diapers were changed. Child refused to lie on the left side. She was first seen at the Children's Hospital at three months. Physical examination revealed nothing abnormal at that time except for slight resistance to motion. Note shallow left acetabulum and slight upward displacement of the femoral head. No treatment instituted. *B.* At six months when there was slight piston mobility. Hip was reduced by closed method at which time a definite click was heard when the head went in the socket. Treatment resulted in a satisfactory hip.

Fig. 8. This film was taken for the lower extremities of a cretin, aged two months. She had the typical flabby musculature but nothing abnormal was suspected in the hips. The part of the film in the region of the hips was, as can be seen, quite light and the subluxation on the left was overlooked. Note how a line drawn along the inferior margin of the os pubis and ischium on the left, when continued, is well below the curve of the neck of the femur. The acetabulum is shallow. The femur is displaced upward (see also Fig. 9).

Now in addition to these, there undoubtedly is an occasional dislocation occurring prenatally, but I know of no reported cases except in association with multiple other deformities. Chandler[2] has reported one such case with multiple congenital contractures of the extremities. As a result of

Fig. 9. *A*. Same case as in Figure 8, a cretin. This patient responded well to thyroid therapy but when beginning to walk at the age of fourteen months a painless limp developed. Note complete dislocation of the left hip. *B*. Same case ten months after treatment by closed method.

these contractures the extremities were maintained in bizarre positions in utero. This is a rare condition, but when present may account for a dislocation truely prenatal in origin.

DIAGNOSIS

A statement frequently made that the roentgen findings are typical and the roentgen diagnosis easy is true only after some time has passed. At birth or immediately thereafter the diagnosis of these infantile dislocations depends, I believe, on a consideration of the factors mentioned above.

In the early post-natal period I do not expect to find any distortion or abnormal shallowness of the acetabulum. The two youngest cases we have encountered, one at eight days and the other at twelve days of age, showed luxation as indicated by the position of the upper end of the femora. Whether this could be demonstrated at birth I do not know. In a relatively short time, two months or less, the upper portion of the acetabulum begins to show flattening as the result of pressure of the misplaced femoral head. The slack in the soft tissues is evidently reduced as muscular activity and tone increase. In the case shown in Figure 1, the physician was easily able to dislocate and reduce the hips immediately after birth, but when the case was seen at eight weeks no abnormal mobility could be detected clinically. No films were obtained at that time as the orthopedist examining the child thought the hips were normal.

The degree of displacement of the femoral head may vary in different individuals at any given age period. In some

Fig. 10. The pelvis and hips of a girl, aged six, with extrophy of the bladder. Note the separated symphysis and wide pelvis. Of 40 such cases reviewed none had dislocated hips. The acetabula were always well formed. This seems to indicate that widening of the pelvis does not explain dislocated hips.

cases it is completely dislocated in a few months (Fig. 4), and in others this may not occur until or even after walking begins (Fig. 11). Some potential cases never dislocate, as has been pointed out.

A line drawn along the inferior border of the rami of the os pubis and ischium, if prolonged outward in the same curve, will fall in line with the inferior surface of the neck of the femur in normal cases. If there is any upward displacement of the femur this line meets the shaft lower down (Figs. 8 and 9).

The head normally ossifies between three and six months of age but in case of dislocation it is delayed. However, delayed ossification of the head does not necessarily mean dislocation, for it is seen occasionally in an otherwise normal hip.

There are times when it is impossible to say definitely whether a hip is or is not going to result in dislocation. In such cases frequent check up examinations may be the only answer to the question. Some surgeons advise mild abduction apparatus in cases which cannot be other than equivocal and in view of the difficulties which may be encountered if treatment is long delayed, abduction "prophylaxis" may be justifiable even before a definite diagnosis can be made.*

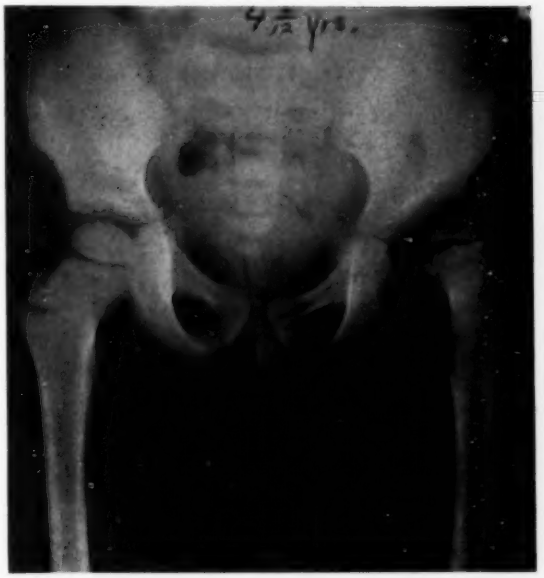

Fig. 11. Hips of a boy, aged four, who had been born at term and walked normally at fourteen months. About a year prior to entry he had fallen down a flight of stairs, following which he developed a left limp. Note that the acetabulum is quite well formed. What had been a potential "congenital" dislocation had evidently been completed by the fall.

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* Since this manuscript was presented for publication, my attention has been called to an article by L. Sedillot, 1835, *2*, 563, in which he stated that he believed that the most frequent cause of congenital dislocation was a softness and relaxation of ligaments.

THE ELIMINATION OF INTESTINAL GAS SHADOWS IN ROENTGENOGRAPHY

PRELIMINARY REPORT ON 1,000 CASES

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ONE of the most important technical factors in the success of a roentgenologist is the clarity of his roentgenograms—images produced without distortion, with no lack of sharpness resulting from motion,

FIG. 1. Case 1. Roentgenogram showing intestinal gas shadows before the use of pitressin.

with no poor contrast or confusing shadows.

It is not the intent of this paper to point out technical factors already well known that enter into the production of good films. Rather, it is our hope that a method will be established to aid in eliminating gas shadows, which, on a roentgenogram, not only cause confusion but also error. This work has been carried out over a period of about one year and includes an observation of more than 1,000 patients.

Since the use of the roentgen ray in the examination of abdominal and pelvic organs, normal and abnormal collections of gas in the bowel have handicapped even the keenest of roentgenologists. Many methods have been utilized in an endeavor to eliminate this source of error. Simple enemata and cathartics, most common of which is probably compound licorice powder, and pressure applied by inflated bags have all met with indifferent success. Not until recently has there been any real advance in the solution of this problem. In this discussion, we wish to present the findings of a year's study of the general results obtained by use of pitressin.

In 1928, Kamm, Aldrich, Grote, Rowe

FIG. 2. Case 1. Roentgenogram taken after the use of the routine procedure outlined. Note disappearance of gas shadows.

and Bugbee first described a method for separating the oxytocic and pressor principles of pituitary extract. The oxytocic element, or alpha-hypophamine, having action on the uterine musculature needs no further mention. However, the pressor substance, "pitressin," through its physiologic action lends itself peculiarly to a subject which has been of interest to every roentgenologist.

The interest in this presentation is the action on the smooth muscle of the intestinal tract. Repeated doses increase the tone of the bowel without ill effect. In our experience no harmful after-effects have been observed.

INDICATIONS

At this point it might be well to enumerate the indications for the use of pitressin, strictly from the roentgenologist's point of view. In general, we know that there is a collection of air or gas in the bowel when the normal peristalsis and churning action are inhibited as a result of obstruction, trauma, infection or nervous imbalance. Also gas is normally present in the colon. Since pitressin acts to produce an increase of muscular tone (with peristalsis) of the bowel, it is apparent that this activity is responsible for the elimination of gas pockets. Its use, we have found, is indicated in any case in which gas shadows obscure findings on the roentgenogram. Some of the uses specifically are in the following:

Fig. 3. Case II. Roentgenogram taken before the use of pitressin.

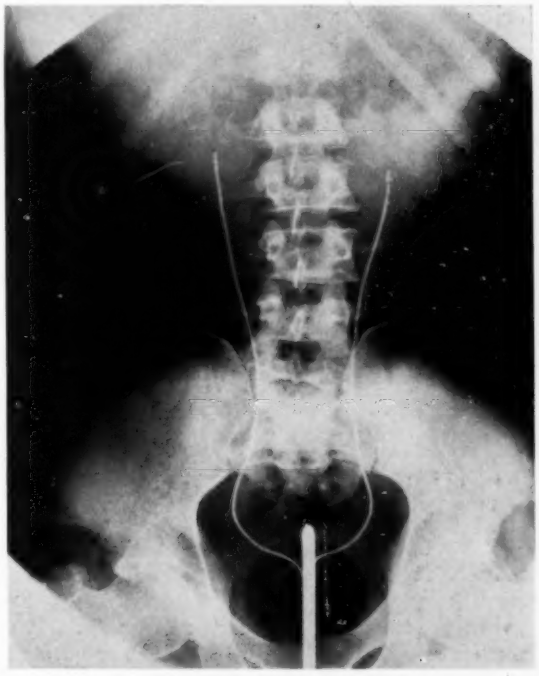

Fig. 4. Case II. Roentgenogram taken after the use of the routine procedure outlined. Note disappearance of gas shadows.

1. Cholecystography—Small gas pockets, in superimposed hepatic flexure, may be misinterpreted as negative shadow stones.
2. Examinations of the kidney, ureter and bladder to reveal the presence of stones, tumors, perinephritic abscess, etc.—The kidney and psoas are often obliterated by gas or fecal matter.
3. Intravenous urography—As the density of the dye is not great, the picture is easily obscured by intestinal gas or feces.

FIG. 5. Case III. Roentgenogram taken before the use of pitressin.

4. Retrograde urography—It has been observed that due to the trauma of cystoscopy and catheterization, a very low grade ileus is produced with resultant gas formation.
5. Examinations of the lumbar spine and pelvis—Gas shadows may simulate bone destruction or metastases.

CONTRAINDICATIONS

The only subjective complaints registered in over 1,000 cases in which pitressin has been used were occasional cramps, sensations of giddiness, weakness, cold perspiration and slight nausea. These symptoms were rarely encountered and were fleeting in character. Objectively, there was noted some blanching, acceleration of the pulse and rise in blood pressure. The greater number of these reactions were encountered in patients who were definitely dehydrated. No lasting effects were observed and it was found that a glass or two of water given at the time of injection minimized the symptoms. Although we have found no absolute contraindications from our own personal experience, extreme care should be exercised in the use of pitressin in cases of cardiac decompensation associated with marked hypertension, coronary sclerosis and thrombosis, and acute, complete mechanical intestinal obstruction of long standing. Pregnancy has not proved to be a contraindication, at least through the sixth month. We encountered no serious reactions, immediate or late, in any of our cases.

METHOD OF ADMINISTRATION

Various time intervals were utilized to determine at which interval the optimum effect in eliminating gas was to be obtained. It has been our experience that the maximum degree of response is obtained approximately thirty to forty-five minutes following injection. It has also been noted that gas shadows reappear in about two to three hours. We have therefore adopted the routine which follows in all cases scheduled in advance:

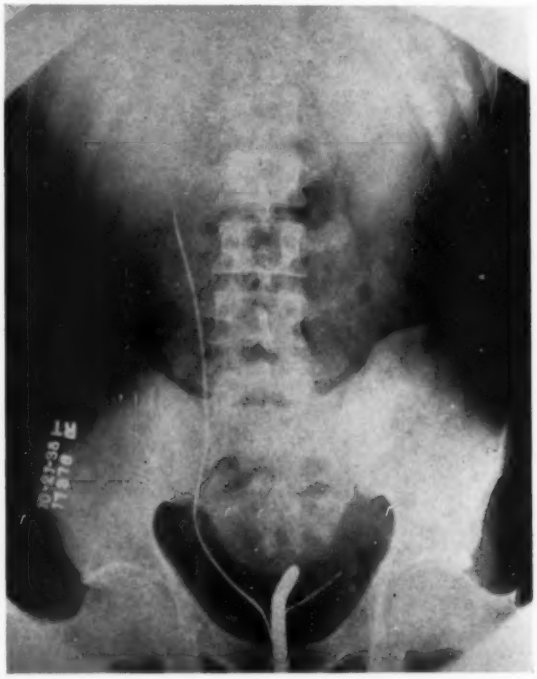

FIG. 6. Case III. Roentgenogram taken after the use of the routine procedure outlined. Note disappearance of gas shadows.

The night before examination a cleansing enema is given.

This is followed in the morning, about one and one-half hours before the time set for roentgenography, by another enema.

One hour before, 0.5 c.c. of pitressin is given intramuscularly. This is repeated in one-half hour.

No cathartic is given.

The enema given in the morning is an aid in clearing the colon of gas shadows. In more stubborn cases where, due to reflex ileus, traumatic ileus, or peritonitis, the cleansing is more difficult, further injections of pitressin may be given. In some few cases of peritonitis and traumatic ileus as many as six ampules (0.5 c.c. each) have been given to produce satisfactory roentgenograms. However, in most cases, two injections of 0.5 c.c. each were found to give the desired results.

We do not mean to imply that there will be no evidence of gas within the bowel; however, it is minimized to such an extent that the roentgenograms are clearer and much more diagnostic than have been obtained by any other method. Also, it has been noted that the hepatic flexure of the colon is shortened, being drawn down away from the gallbladder area, thus eliminating shadows due to superimposed bowel. This, we feel, is of inestimable value.

It should be stated that, with but few exceptions, our investigation was confined to hospitalized patients. This was done for several reasons. First, because the presence of intestinal gas is more abundant in recumbent patients, whereas ambulatory cases seem to maintain a more normal physiologic activity of the bowel. Thus, there is less opportunity for gas to collect. Secondly, nearly all urographic examinations are made on in-patients. Also in handling a large number of out-patients, our facilities are taxed to such an extent that unnecessary delays must be avoided. However, the routine may be adopted on these cases after more careful evaluation of the results of our studies.

In regard to the expense of this method, we believe that the cost of the medication has been amply compensated for by the saving in time, re-examination and the greater ease in diagnosis when better quality films are available.

We would like to mention several rather striking examples of the successful use of pitressin. One case, although not a problem in diagnosis, illustrates the action of pitressin very well. A female, aged thirty-five, three months pregnant, presented a history of very obstinate constipation. Two to three weeks would pass without bowel action. A barium enema was given with fully 2 quarts of barium necessary to fill the colon, which was markedly atonic. The patient was unable to evacuate any of the contents of the enema in spite of repeated effort. It was decided to give one ampule of pitressin. The patient evacuated the enema, as well as a very large amount of fecal matter, fifteen minutes after injection. (Pitressin is being studied in atonic colon and megacolon.)

Another patient with suspected fracture of the kidney was examined by both retrograde and intravenous methods on three occasions. All films taken were non-diagnostic due to the gas in the bowel secondary to a traumatic ileus. Pitressin was given as outlined and intravenous urography again done, with excellent results. Numerous other cases equally noteworthy were encountered, but we shall not present them in this preliminary report. We are including a few representative films to illustrate the efficacy of our method.

SUMMARY

1. A new method of eliminating gas shadows in roentgenography of the abdomen is presented.

2. The "pressor" principle of pituitary extract, pitressin, is the drug of choice.

3. Indications and contraindications are noted.

4. A simple method of administration is given.

5. Representative roentgenograms are shown.

CONCLUSIONS

We believe that this method is of definite advantage in roentgenography, and that it can be used as a general routine in any hospital. We shall subsequently present more detailed data.

We wish to express our appreciation to the visiting and resident staff of Receiving Hospital in the various departments, especially the Genito-Urinary Service, for their cooperation.

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Vol. 37, No. 1

PANCREATIC LITHIASIS

REPORT OF CASE*

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THE case presented is of interest chiefly because of roentgenographic findings. Cardiac complications in this case are considered too severe to warrant surgical intervention.

The rarity of pancreatic calculi is stressed by all authors. Since Ackman and Ross collected 108 cases, including one of their own in 1932, five additional cases have been reported in the literature (Collins, Rienhoff and Lewis, Thomason, and Bost, 2 cases). Of this total of 113 cases, 79 have been post-mortem reports. In only 4 of the remaining cases was roentgen diagnosis made prior to surgery (Lindsay, Thomason, Collins and Bost). Pancreatic calculi have been more frequently observed on the roentgenogram but were considered to be gallstones or kidney stones and the proper diagnosis was made only after surgery for these conditions. In several cases the pancreatic stones were palpated but not removed during laparotomy and were later demonstrated roentgenographically in postoperative studies.

The admirable papers of Bost, and Ackman and Ross cover the matter of surgical treatment and symptomatology quite adequately and the reader is referred to these for full details. It is sufficient to say that surgical treatment is feasible, the mortality from surgery being about 7 per cent. The symptomatology is variable. Pain is the most constant sign. In 19 of 29 surgical cases collected by Ackman and Ross epigastric colic was noted. They also noted radiation of pain to the thoracic region of the back, to the left lumbar region and to both lumbar regions. In only 2 of these cases was pain reported as absent. Dull epigastric pain, nausea, vomiting, late glycosuria, jaundice, diarrhea, stones in the stool, marked loss of weight, skin pigmentation, steatorrhea, palpable tumor and history of duodenal ulcer were mentioned, but none of these signs were present in sufficiently large proportion to be diagnostically significant. The etiology is obscure. Seeger reported that pancreatic calculi contain calcium carbonate but that normal pancreatic juice contains no calcium carbonate and concluded that altered secretion precedes stone formation in the pancreas.

FIG. 1. Roentgenogram showing multiple pancreatic calculi.

* Published with the permission of the Medical Director of the Veterans' Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the writer.

FIG. 2. Semi-lateral view showing multiple pancreatic calculi.

It is to be presumed that the reported cases represent only a small proportion of such cases. Opie found 2 cases in 1,500 autopsies. This would indicate that while the condition is relatively uncommon, it is not rare.

The question of why pancreatic stones are demonstrated so infrequently on roentgenograms arises. A possible explanation may be found in the anatomical relationship of the pancreas to the stomach and intestines. With opaque medium in the stomach and intestines the site of the pancreas is almost totally masked in the usual gastrointestinal study. In the case reported here the stones would have been overlooked entirely had they not extended to the tail of the pancreas and projected slightly above the level of the stomach. It is rather unfortunate that the relative infrequency of the condition does not warrant the routine exposure of a film before the administration of the opaque meal. Roentgenoscopy does not offer a solution. In spite of the large number of stones, their size and relative density and a prior knowledge of their existence, the pancreatic stones in our case could not be visualized roentgenoscopically.

REPORT OF CASE

A white male, aged forty-five, was admitted to the Veterans' Administration Facility, Dwight, Illinois, January 18, 1936, for the treatment of hemorrhoids and undetermined stomach condition. In 1922 appendectomy and cholecystectomy were performed. Prior to that time and up to about 1933 the patient had vague, indefinite but constant symptoms which can best be described as dyspeptic. In 1933 these symptoms became more bothersome and were accompanied by dull pain in the epigastrium with occasional colicky radiations to both lumbar regions. Lately the epigastric pains have become more severe and the colicky radiations more frequent. Patient has been constipated for many years and bleeding from the hemorrhoids has been moderate. Symptoms

FIG. 3. Roentgenogram showing partial masking of the pancreatic calculi by the opaque meal.

of moderate cardiac impairment were elicited. There has been considerable nausea but no vomiting. There has been no jaundice and weight has remained constant since discharge from World War service. Steatorrhea is not present.

On examination patient was found to have mitral heart disease, hypertension, left inguinal hernia, well-healed appendectomy and cholecystectomy scars, moderate hemorrhoids and a quiescent anal fistula. There was moderate tenderness in the epigastrium but no masses were palpable.

The Wassermann reaction was negative. The blood and urine studies were within normal limits. Stool examination revealed an unusually large amount of undigested meat fibers. The Brown test for amylase in the feces and the Gross test for trypsin in the feces indicated marked diminution but not entire absence of these pancreatic ferments. Roentgen studies revealed multiple calculi along the entire length of the pancreas.

The patient is being retained at this Facility for treatment of the heart condition. Surgical treatment for the pancreatic calculi is not contemplated.

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GIANT CELL TUMORS OF BONE

EXPERIENCE WITH SURGICAL AND ROENTGEN TREATMENTS ON A MATERIAL OF FIFTEEN CASES

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IN A RECENT paper[1] we reviewed the cases of non-generalized osteitis fibrosa as they came to observation in the Departments of General and Orthopedic Surgery of the State University of Iowa. We arrived at the conclusion that there are different forms of localized osteodystrophy. There are localized cases of a solid (pseudocyst) or a cystic (true bone cyst) type, usually situated in the upper portion of the diaphysis of the humerus, femur and tibia; less frequently diaphyseal localization is observed; never localization in the epiphyses. Besides the localized form, more extensive cases can occasionally be seen with involvement of the greater part of the diaphyses. Multiple localized lesions are rare but may occur without any apparent rule in the distribution of the lesion. There is also a strictly unilateral (monomelic) form of fibrous osteodystrophy with involvement of the entire or of great portions of the diaphyses of bones, arranged in longitudinal ray-like anatomic distribution. On the basis of such observation we believe that the localized form represents an abortive type of the extensive monosseous form. The latter is to be considered as an abortive form of the monomelic type which, in turn, is most likely an abortive form of the unilateral osteodystrophy. Inasmuch as the latter cannot be taken as traumatic in origin, but must be due to an endogenous cause entirely unknown, the purely traumatic etiology of all the other forms is rather doubtful.

We did not include in that paper the cases of giant cell tumors which, although they are quite commonly considered as localized forms of osteitis fibrosa, act clinically at least in many respects quite differently. We were especially interested in the question of treatment and of recurrence following roentgen or surgical therapy. In this paper we shall review 15 cases, 4 males, 11 females, the ages ranging from eight to sixty years, average twenty-seven years.

First decade of life 1 case
Second decade of life 3 cases
Third decade of life 8 cases
Fourth decade of life 1 case
Fifth decade of life 1 case
Sixth decade of life 1 case

ANALYSIS ACCORDING TO LOCALIZATION

1. Lower End of Radius—3 Cases

CASE I. Woman, aged twenty-seven, who about one year before admission, a short time after a fall on the extended right hand, began to have pain in the right arm and a swelling appeared in the right wrist region. Five months before admission she sought treatment in a hospital in Des Moines. A cyst was opened in the lower end of the radius and cleaned out. Histologically no signs of malignancy were found. Following this operation she was free from pain until two weeks before admission to our clinic. At that time swelling had reappeared at the same place. Roentgenograms showed a multilocular cyst at the lower end of the radius. A second operation was suggested to the patient but was refused. No further note could be obtained.

CASE II. Male, colored, aged thirty-six. About one year ago patient first noticed a dull ache in the right wrist. A few weeks later he fell upon the outstretched hand. Roentgenograms were taken and a pathological fracture in a bony tumor was found. Cast was applied for seven weeks and then he went back to work with apparently good use of the wrist. Six months ago he had a direct trauma to the right wrist, resulting in fracture. Cast for six weeks.

[1] Freund, E., and Meffert, C. B. On different forms of non-generalized fibrous osteodystrophy. *Surg., Gynec. & Obst.*, 1936, *62*, 541–561.

* Florida Medical Center, Venice, Florida.

Since that time limitation of motion and increase of size of tumor.

Roentgenograms showed a very extensive destructive lesion of the lower end of the radius which was replaced by a loose sponge-like mass of bony tissue with fairly good outline at the volar side, but extensive invasion of soft tissues on the dorsal side. Very marked soft tissue swelling. The tumor extended clear down to the wrist joint. The subchondral bony lamella of the radius was broken through in several places.

Case III. Woman, aged twenty-three, who fractured the lower end of the right radius six months ago. Fracture healed within six weeks but wrist remained swollen. Four months after the fracture a small mass appeared at the site of callus, which since has steadily increased in size. Roentgenograms showed a destructive lesion of the lower end of the radius, extending down to the subchondral bony lamella and, after destruction of the cortical bone, growing extensively toward the surrounding soft tissue.

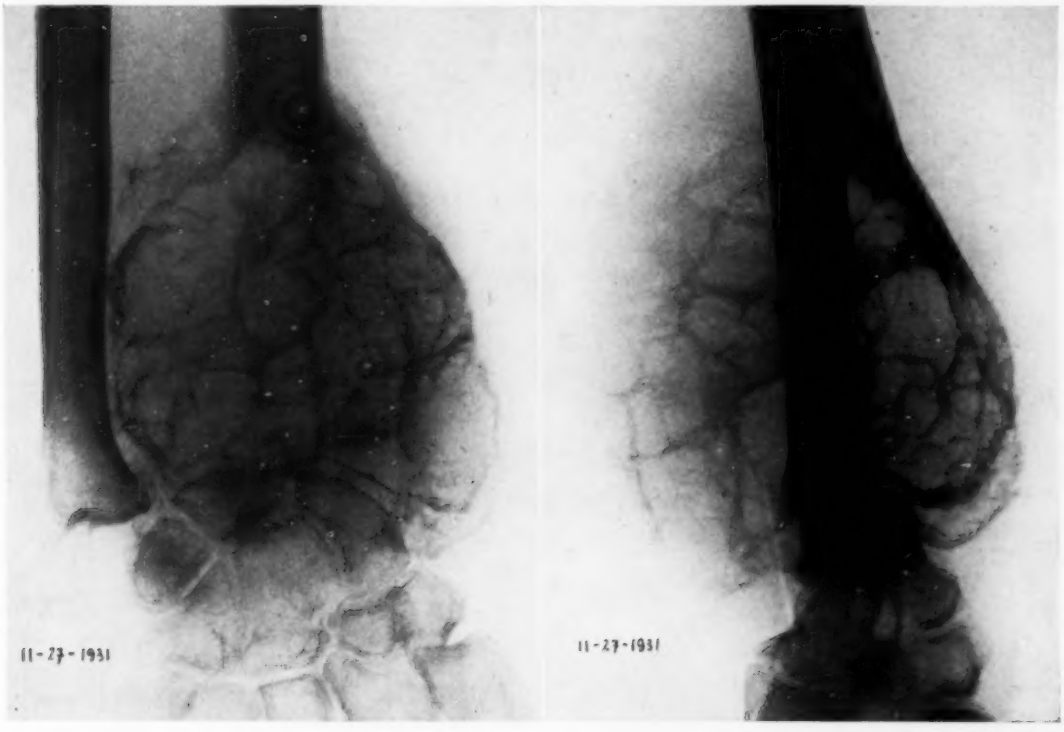

Fig. 1. Case 1. Female, aged twenty-seven, with extensive giant cell tumor of lower end of radius. Recurrence after curettage.

Patient received roentgen treatment to the tumor without any improvement. Therefore, surgical treatment was decided upon.

Operation. A very thin shell of bone was broken through at several places by brownish tumor tissue which was well defined in a capsule. The bony shell was opened and semi-solid and dark brown tumor tissue was encountered. The cavity resulting after removal of the tumor tissue was the size of a goose egg. The joint cartilage was bulging but not broken through. The whole content of the cavity was carefully removed by curette.

Pathology Report. Giant cell tumor. No follow-up since operation.

The tumor mass was, however, still well outlined and in many places surrounded by a thin bony shell.

Because of the proximity of the tumor to the wrist joint, it seemed that surgery was contraindicated and roentgen therapy was instituted. Despite the roentgen therapy the tumor showed very rapid increase in size and so operation was performed.

Operation. (Nine months after first admission.) The tumor had invaded the subcutaneous tissue of the dorsum of the hand very extensively. In some portions, however, a fairly satisfactory removal of tumor tissue could be carried out. Most of the extensor tendons passed

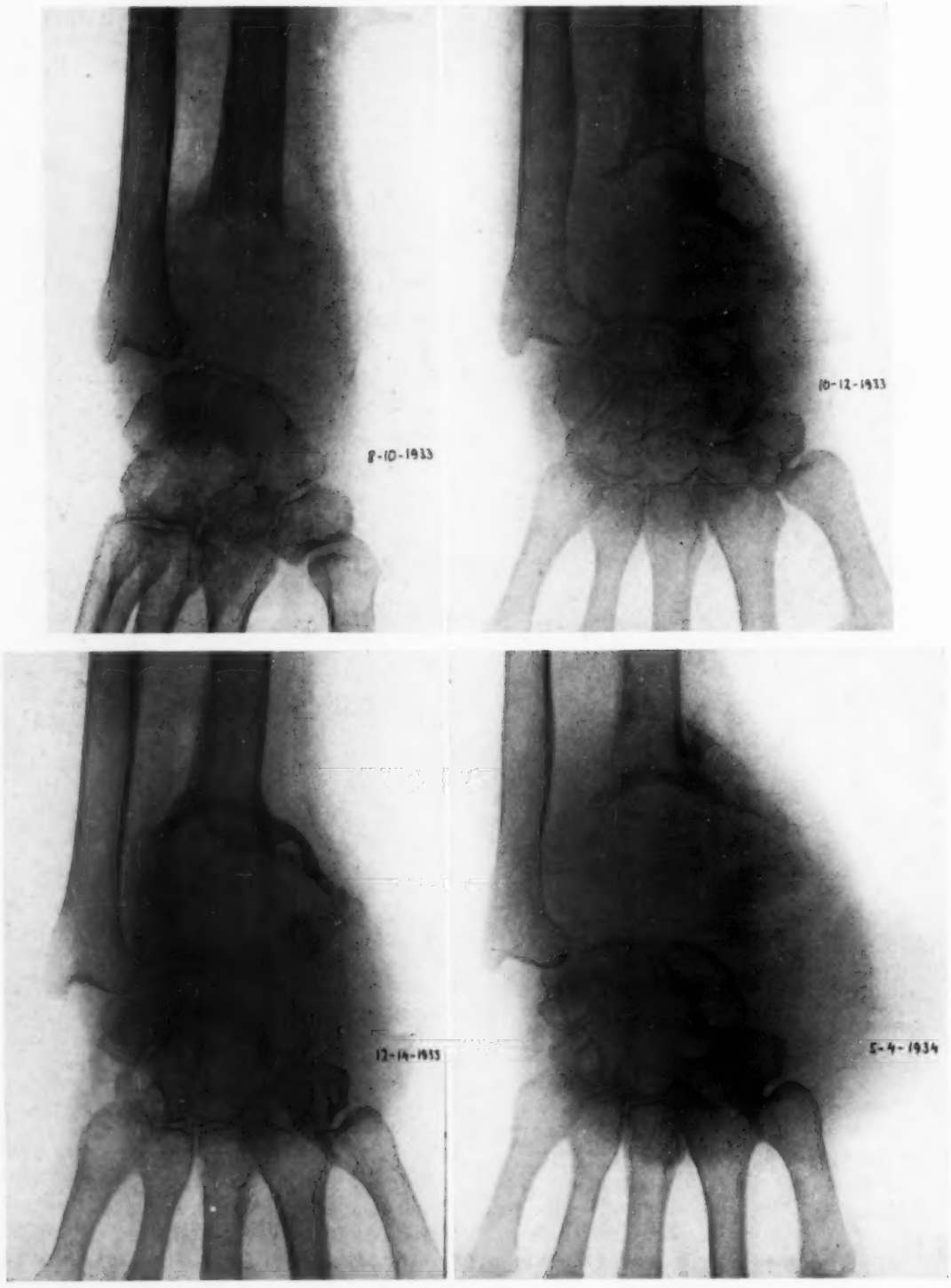

FIG. 2. Case III. Female, aged twenty-three. Very extensive giant cell tumor of lower end of radius, involving the wrist joint. Progressive despite roentgen treatment.

directly through the tumor. The articular surface of the radius was represented by very thin tissue which was partly broken through by the tumor. The joint capsule was invaded. The whole tumor mass was as large as two fists, with softening of the tissue in some places. Be-

cause of the extensive involvement of the soft tissues in the wrist joint, amputation was performed.

Pathology Report. Giant cell tumor.

2. Lower End of Humerus—1 Case

Case iv. Boy, aged eight, who fell six weeks before admission and heard his arm crack. Since then, slight pain in the arm near the elbow, and the bone has gradually grown to the size of a goose egg. Roentgenograms showed a localized cystic lesion in the lateral condyle of the humerus, with enlargement of the bone.

Operation. Very thin shell of bone covering a dark blue tumor tissue, containing a small cystic cavity. Careful curettage.

Pathology Report. Giant cell tumor.

Following operation there was some drainage for about three weeks. No pain. Six months later, definite clinical and roentgenologic signs of a local recurrence. Second operation: Another attempt at radical curettage was made. The resulting cavity was cleaned out with 95 per cent phenol and alcohol. No follow-up after second operation.

3. Upper End of Ulna—1 Case

Case v. Woman, aged thirty, who has had for the last ten years swelling of the upper and medial aspects of the left forearm. This has become tender during the last few years. On physical examination a swelling of bony consistency was found in the right ulna below the elbow which, however, did not interfere with motion. Roentgenograms showed an extensive multilocular lesion in the upper half of the ulna, leading to distention of the bone and thinning of the cortex.

Patient was submitted to roentgen treatment which was carried out more or less at monthly intervals over a period of fifteen months. The roentgenologic follow-up of the case showed a steady and definite improvement with good calcification and healing of the lesion.

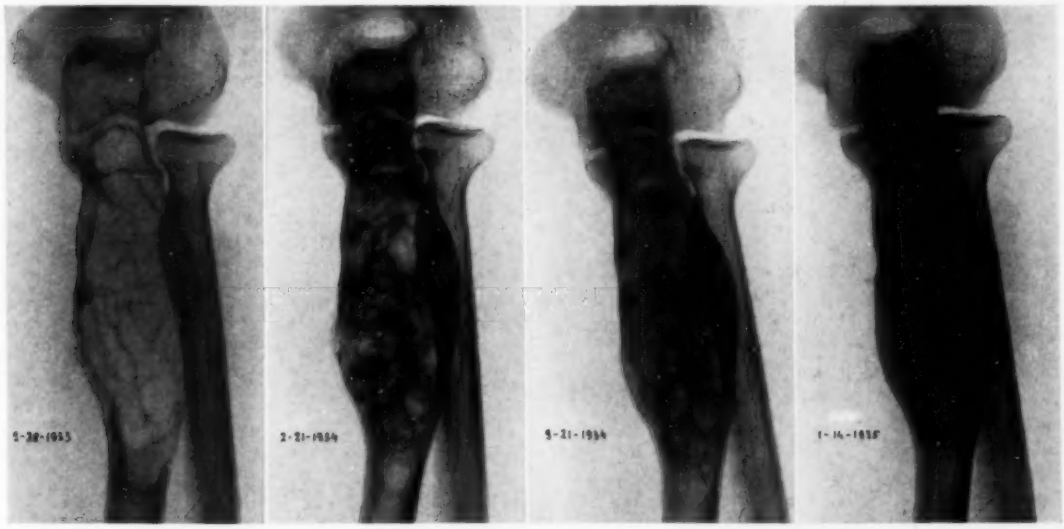

Fig. 3. Case v. Female, aged thirty. Giant cell tumor of upper end of ulna. Gradual healing under roentgen therapy.

4. Lower End of Ulna—1 Case

Case vi. Male, aged sixty, with retroperitoneal tumor. One year ago he noticed slight pain in the left wrist. This became gradually worse and finally he noticed a small tumor in the wrist region. Roentgenogram showed a cystic porotic area in the lower end of the ulna, with thinning and interruption of the cortex, but apparently without soft tissue involvement. The lesion was very suggestive of giant cell tumor. No treatment.

5. Lower End of Femur—4 Cases

Case vii. Female, aged twenty-one. Two years before admission patient had a bone cyst curetted in the left knee region. There was a mild postoperative infection with some discharge. Immobilization in cast and brace for almost two years. Weight-bearing for the last six months. Swelling reappeared three months ago. Roentgenograms showed a large cystic area in and above the outer condyle of the femur, well surrounded by an irregular zone of osteosclerosis. The cortex over the lesion was expanded but not thinned out.

Patient received a few roentgen treatments but left the hospital before evidence of any changes.

CASE VIII. Female, aged twenty-four, who three years before admission, following a fall, suffered severe pain in the left knee. Weight-bearing impossible. Roentgenograms taken at that time were supposedly negative. Since then, frequent attacks of pain and swelling in the knee region.

Roentgenograms showed an extensive cystic lesion in the medial condyle of the femur, extending upward in the lower third of the diaphysis and also in the lateral condyle. The bone at the site of the lesion appeared blown up, the cortex was thinned out but apparently not interrupted.

Operation. A multilocular cystic tumor was found with walls of soft tumor tissue of dark brown or red color. Careful curettage. A bone graft was removed from the left tibia, broken up and inserted into the cavity which was about 4×6 inches in size. Sterile vaseline to fill the cavity. Uneventful recovery.

Pathology Report. Giant cell tumor.

Roentgenologic follow-up three months after operation showed that the grafts had taken and there was good osteogenesis around them. Nine months postoperatively a very large cavity could still be seen and fourteen months postoperatively one gained the impression of a definite progression of the lesion in the lateral condyle. Clinical examination, however, three and five years after operation, showed almost perfect motion in the knee. No pain. Brace discarded.

CASE IX. Male, aged forty-eight, with pain in the lateral side of the right knee for one year. Patient never stopped working until two days ago, when he slipped, striking his right knee. Roentgenograms showed a typical picture of giant cell tumor in the lateral condyle of the femur, extending down to the joint surface. There was an intra-articular fracture through the lateral condyle, which was slightly displaced backward. No callus formation.

Patient received roentgen treatment and was immobilized in a plaster cast. Despite repeated roentgen treatments, the lesion showed roentgenologically definite signs of progression. Surgical treatment was therefore indicated.

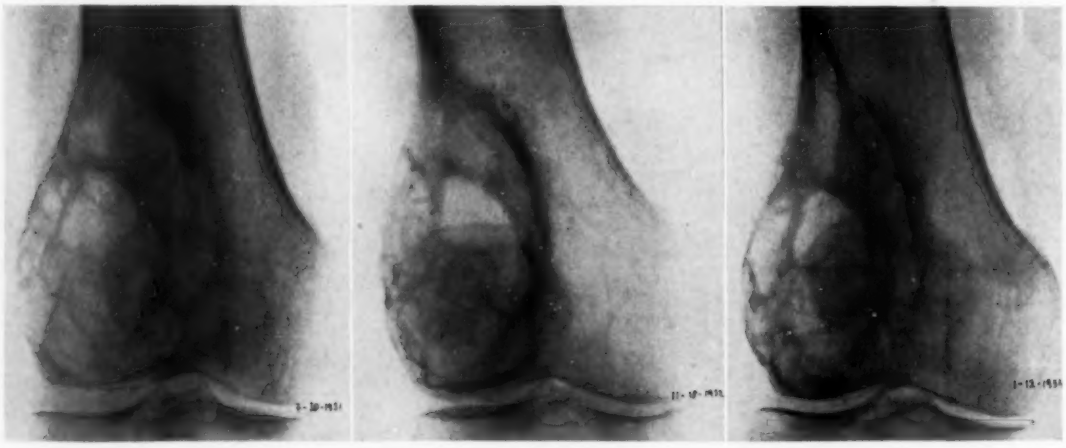

Fig. 4. Case VII. Female, aged twenty-one. Giant cell tumor of lower end of femur, treated by curettage, followed by slight infection.

Operation. (One year after first admission.) A window was cut in the very thin shell of bone and a very large cyst was opened. It occupied all of the external condyle and extended up into the shaft and down to the joint cartilage. Careful curetting was done. The wound was washed with iodine, alcohol and saline.

Pathology Report. Giant cell tumor with cyst formation.

Following operation there was no tendency to healing. The large cystic area remained unchanged. Patient was submitted to intensive roentgen treatments for a period of eight months. No result. Therefore, it was suggested to the patient to have either another attempt at curettage followed by bone transplantation, or amputation. Neither suggestion was accepted.

CASE X. Female, aged eighteen. Three years ago patient fell and sustained a lateral dislocation of the left knee, which her home doctor attributed to a weakened condition of the bone.

Cast for two and one-half months. Since then, limitation of motion in knee joint with swelling. Pain only at night. On physical examination the lateral condyle of the femur was found blown up and formed a mass of hard bony consistency. Roentgenograms showed a big cystic area in the lateral condyle of the femur, leading to expansion of the bone but without breaking through of the cortex. Marked valgus deformity of the knee joint.

Operation. Under tourniquet the lateral condyle was exposed. The periosteal surface was smooth; no defects, but the cortex extremely thinned. Almost the entire condyle was filled with solid tumor tissue, brownish gray with yellow areas. The central portion showed beginning cyst formation. Careful curetting into healthy bone tissue. Many small bone chips were removed from the tibia and the bone cavity was filled to about three-fourths.

Pathology Report. Giant cell tumor. Uneventful recovery. Cast, followed by long leg brace.

Roentgenograms four, seven and eleven months after operation showed that practically all the bone chips had taken and had led to a very good osteogenesis with almost complete filling of the cavity. No pain, brace discarded.

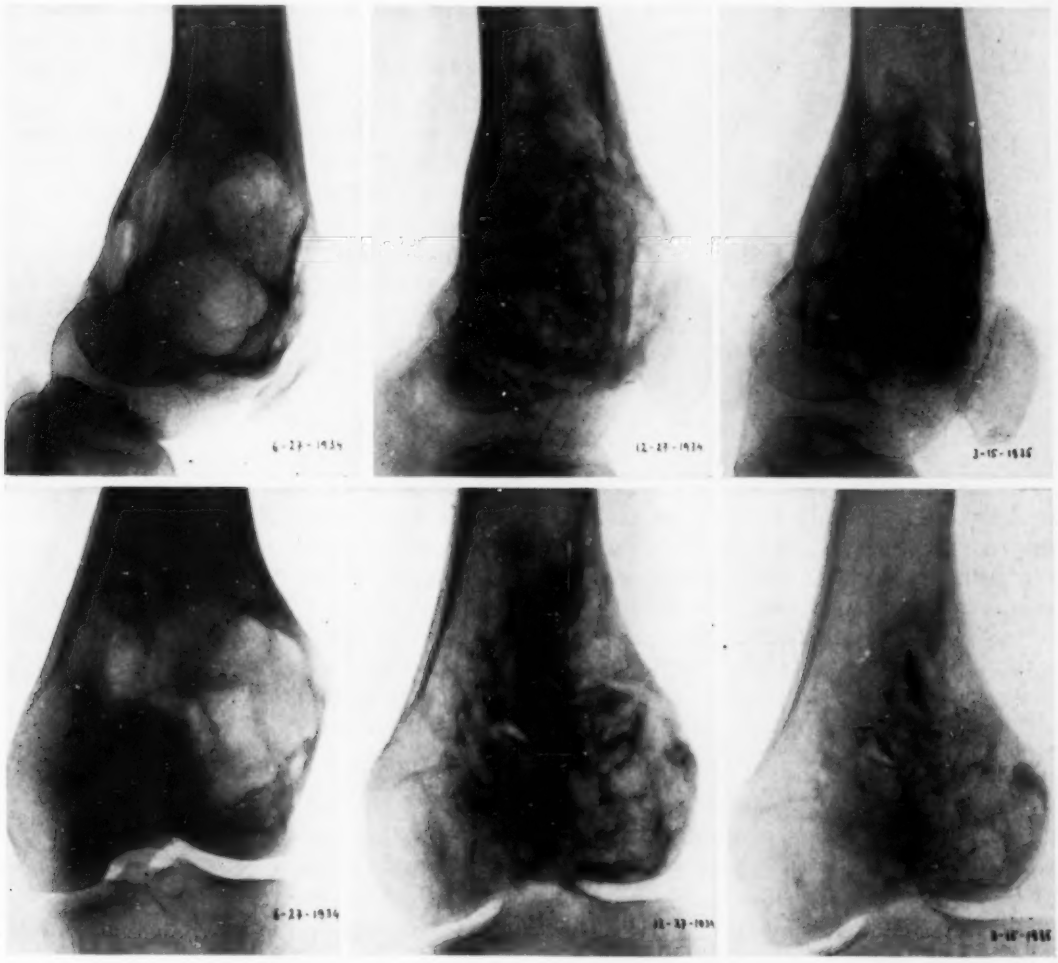

Fig. 5. Case x. Female, aged eighteen. Giant cell tumor of lower end of femur, treated by curettage and bone chips. Nice healing.

6. *Lower End of Tibia—2 Cases*

Case xi. Female, aged twenty, with swelling and weakness of left ankle for the last six months. Roentgenograms showed a destructive lesion the size of a hen's egg in the lower end of the tibia, with a thinning out of the lateral and posterior cortex to a paper-thin shell and extending down to the subchondral bony la-

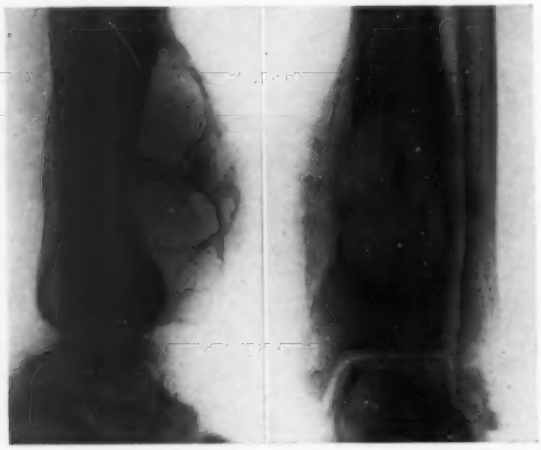

FIG. 6. Case XII. Female, aged sixteen. Extensive giant cell tumor of lower end of tibia, with marked periosteal reaction.

mella of the ankle joint. Some periosteal reaction over the anterior cortex. Soft tissue swelling. No sharp outlines against surrounding soft tissues.

Sarcoma was strongly considered in this case and the patient submitted to three series of roentgen treatments. Clinically, the swelling became smaller and the ankle region was less painful. Amputation seemed to be indicated.

Operation. A fairly well-encapsulated tumor was found, quite fibrous, and the surgeon's impression was sarcoma. Frozen section was studied and reported as sarcomatous. Preparations were made for amputation when the pathologist reported giant cell tumor. Very thorough curetting of the cavity was performed.

Very good recovery; patient gained 18 pounds. Roentgenogram five months postoperatively revealed some reparative changes but also still the characteristic picture of giant cell tumor. No further note.

CASE XII. Female, aged sixteen, with increasing pain and swelling of left leg for last five months. She had been operated on for bone cyst. She made a good recovery until two months ago, when the tumor began to grow again. It grew rather fast. A piece of tissue was removed and examined by a pathologist who pronounced it sarcoma. Roentgenograms showed a cystic area the size of a hen's egg in the lower third of the tibia without sharp outlines. The cortex was thinned by erosion from within and extensively removed at the anterior and inner portions. Especially anteriorly the bone was blown up, showing several cystic areas which were not always well outlined toward the soft tissue. There was definite soft tissue involvement. Higher up and on the inner side the periosteum showed bone production with onion peel like appearance. The joint was not involved.

On the basis of the pathology report the leg was amputated. New sections did not confirm the diagnosis of sarcoma but revealed a rather advanced stage of healing giant cell tumor.

CASE XIII. Female, aged twenty-four, with stiffness of the muscles just below the left knee for eighteen months. Eight months ago, operation for a cyst of the fibula. The tumor had recurred since operation and was now slightly larger. Pathology report was giant cell tumor. Roentgenograms revealed an extensive multilocular cystic tumor in the upper end of the fibula, surrounded by a very thin shell of bony tissue.

Operation. Delivery of tumor was difficult. It reached up into the popliteal space. After careful dissection the entire tumor mass was removed and the upper end of the fibula cut off in healthy tissue about one-half inch below the lower pole of the tumor.

Pathology Report. Giant cell tumor. Uneventful recovery.

About one year following operation, swelling in the outer and upper parts of the popliteal space reappeared. This area was opened and four tumors were removed. They were intimately connected with the sheath of the tibial nerve. There was no connection between them and the bone. Histologically, they showed the typical picture of giant cell tumor. Following the last operation patient had several roentgen treatments. No further trouble. No recurrence. Observation period of almost five years.

CASE XIV. Female, aged twenty-three, with tenderness over the head of the left fibula for about one year. Six to eight weeks before admission this area became very painful, especially on weight bearing. Roentgenograms revealed a purely bone destructive lesion at the upper end of the fibula with expansion of the bone and formation of a very thin bony shell on the surface but not clear around the tumor. No sunray structure. Some bone formation by the elevated periosteum.

Operation. Careful curettage of the tumor mass. Iodine tincture and tight closure.

Pathology Report. Giant cell tumor. One year later, perfectly all right. No signs of recurrence.

Case xv. Female, aged twenty-five. At age of seventeen, shot gun charge into the right knee region. Pain on walking. Two years ago, pain increased. Operation was performed and tumor found in the upper end of the tibia. Diagnosis was giant cell tumor. Bone became infected following the operation and drained for about ten months. She fractured the tibia two months ago, just below the knee. In cast since. Roentgenograms showed a destructive lesion in the upper end of the tibia, extending to the joint surfaces of both condyles. Pathologic fracture through the head of the tibia with some periosteal callus formation. Fracture healed well by immobilization and plaster cast. No further note.

If we now consider our 15 cases of giant cell tumors as a whole and if we try to bring them into relation with the cases of localized osteitis fibrosa, we find certain common symptoms which have led to the widespread opinion that localized osteitis fibrosa and giant cell tumors represent one etiologic entity, but there are also differences which make such an intimate correlation rather doubtful. Both have in common localization of the lesion in the ends of the long tubular bones and a relatively benign clinical course. As a rule, they affect young individuals. Pathologic fractures as early clinical symptoms are very frequent and certain pathologic-anatomic features are the same in both groups. The tendency to cyst formation especially, from the roentgenologic viewpoint, is a quality which renders differential diagnosis difficult. So it is understandable that there are unitarians, especially among the German authors, who consider localized osteitis fibrosa, bone cyst and giant cell tumor as only different morphologic manifestations of the same pathologic process. There are, however, certain features which make it hard to believe that both lesions are etiologically identical. If it were only the process of organization of a medullary hematoma, as it is considered by German authors, then we would expect to find cyst formation later, giant cell tumor first. This, however, is true only to a very moderate degree. There is no doubt that a number of bone cysts are derived from cystic degeneration of giant cell tumors. On the other hand, it is a fact that true bone cysts (not all those from a roentgenologic viewpoint which quite frequently are only pseudocysts filled with some radiotranslucent soft tissue) come to clinical observation at a much younger age (first to second decade of life) than giant cell tumors (late second or third decade of life), and for this reason alone it is hard to conceive that both lesions should be essentially the same. Another difference lies in the localization. We pointed out for the fibrous osteodystrophy in all its different forms that localization in the metaphyses or diaphyses of certain bones is very characteristic. It seems that the epiphyses never become involved. Giant cell tumors, on the contrary, show predilection for the epiphyses. Metaphysis may become affected by continuity but the process apparently starts primarily in the epiphyses close to the joints. There is also a difference as far as the skeletal topography of the affections is concerned. For localized osteitis fibrosa the upper metaphyses of the humerus, femur and tibia are the most frequent sites, whereas giant cell tumors develop most frequently in the lower end of the radius and the femur. These are differences which can hardly be explained on the same etiologic basis. They point to essentially different diseases.

Moreover, the clinical course in cases of giant cell tumors and their response to therapeutic measures differ from the common cases of osteitis fibrosa.

As far as the importance of trauma and fracture in the group of giant cell tumors is concerned, it is much easier to rule out more severe fracture as a causative factor than it is in the group of osteitis fibrosa. None of the cases showed development of giant cell tumor following definite trauma. The onset, as a rule, was very insidious. Fractures occurred only after more advanced destruction and apparently not as frequently as in localized osteitis fibrosa. In our group of 15 cases pathologic fractures were present in only 4 cases (in 2 cases

twice). In one case the fracture was incomplete. So we have to conclude that if it is not unlikely that trauma is a contributory etiologic factor in a relatively small number of cases of osteitis fibrosa, it has but secondary significance in giant cell tumors.

As to the question of treatment and healing tendency, it is quite apparent that osteitis fibrosa is altogether more benign and even shows tendency to spontaneous healing. Only a relatively small number of cases are progressive and tend to recur after operation. Giant cell tumor is by far a more serious bony lesion which, although not malignant from a strictly pathologic viewpoint, certainly acts in quite a number of cases like a tumor with local malignancy from the clinical viewpoint.

So we find that out of 12 cases treated surgically, only 2 (Cases X and XIV) showed definite improvement by the operation. One case (VIII) was benefited clinically but the roentgenograms showed persistence of the large cystic area fourteen months after operation. Case X was not checked up by roentgenograms; the clinical findings, however, were very satisfactory almost two years after operation. Case XIV, with one year of observation, showed a very good result, clinically and roentgenologically, with good osteogenesis around the transplanted numerous bone chips.

All the other cases with surgical treatment—8 in all (in 2 cases there was no follow-up whatsoever since the operation)—are not very satisfactory. We have already mentioned Case VIII which five years after operation (curettage and bone grafts) showed definite clinical improvement; but the roentgenograms in this case still revealed a large bony cavity which was even progressive on one side at last examination. Two cases (Cases IX and XV) although without evidence of progression, did not reveal any healing tendency following curettage. In fact, in one case amputation was considered strongly and in the other a second operation was proposed to the patient.

Five cases (Cases I, IV, VII, XII and XIII) showed recurrence of the tumor usually quite a short time after the operation. One case was operated on for the second time (Case IV) but there is no follow-up since the second operation. Case XIII had to be operated upon three times because of recurrence, and healing did occur only after roentgen treatment was added to the third operation. In Case XII a lesion of the lower end of the tibia recurred after excochleation. Biopsy was pronounced as sarcoma by a pathologist and the leg was amputated. The 2 remaining cases (Cases I and VII) did not accept any further treatment.

It appears thus clearly that the results of surgical treatment in giant cell tumors are far less satisfactory than in localized osteitis fibrosa. It seems, however, that the best chances for surgical interference are given by very radical removal of the tumor into healthy bone tissue, followed by filling in of the cavity with numerous bone chips. One gains the impression that the small chips represent a better stimulus for osteogenesis than larger bone grafts. In cases of very large lesions it may become necessary to remove sufficient bone material from both tibiae to fill the defect resulting after operation. In this respect, there is no difference between giant cell tumor and bony defects due to curettage of localized osteitis fibrosa.

Giant cell tumors are commonly considered as being very susceptible to roentgen treatment. In our series we have only 5 cases in which roentgen irradiation was used as a therapeutic measure. In 2 cases (V and XI) there was very good response. In Case V (roentgen treatment for about one and one-half years) there was definite improvement of the lesion, although no return to normal bony structure. In Case XI the tumor became smaller and the patient was then submitted to operation. In another case (Case XIII) in which three operations were performed previous to roentgen treatment, this treatment secured apparently permanent cure. Two other cases (Cases II and III) did not respond at all to roent-

gen irradiation and were therefore submitted to surgery. Both cases were of pronounced clinical local malignancy.

As far as these 5 cases permit, we conclude that some cases of giant cell tumors may respond well to roentgen treatment; others, again, do not react favorably and may even increase in size, despite or possibly because of the roentgen effect. Further observations and a study of a greater material will be necessary to arrive at more definite conclusions.

There can, however, be no doubt that quite a number of giant cell tumors, at least from the clinical viewpoint, remain on the borderline between benignity and malignancy. It is true that they do not form metastases, and with amputation it seems that the lesion becomes definitely eradicated; but the local findings frequently show marked destruction with invasion of the surrounding soft tissue. Such cases as II, III, XI and XII in our series showed very extensive involvement of the surrounding soft tissue so that in 2 cases amputation was performed and in the 2 other cases it was very strongly considered. We have good reason to believe that even if benign from a strictly pathologic viewpoint, a goodly number of giant cell tumors certainly do act like local malignant tumors from a clinical viewpoint. If roentgen treatment is of no benefit in these cases and surgery faces the impossibility of radical removal, amputation still seems to be the only and quickest way to secure permanent cure. Such "borderline" cases are relatively frequent among giant cell tumors; infrequent in the group of osteitis fibrosa. Of the latter, only the solid, not the true cystic form shows tendency to progression and recurrence. We had in our series of 25 cases of osteitis fibrosa only one case, a diaphyseal lesion in a very young child, which resembled very much "malignant giant cell tumor" from a clinical viewpoint. The lesion recurred very rapidly following the first operation and a second radical curettage was performed. Unfortunately, the case could not be followed up after the second operation.

Summarizing our experience on a material of 40 cases (15 giant cell tumors, 25 osteitis fibrosa), we can state that, despite several factors which they have in common, localized osteitis fibrosa (solid or cystic form) has to be considered as different from giant cell tumor, at least clinically. *Age* (giant cell tumors, second and third decades of life; localized osteitis fibrosa, first and early part of second decades of life); *localization* (giant cell tumor mainly in the lower epiphyses of the radius and femur; osteitis fibrosa in upper metaphyses and diaphyses of humerus, femur and tibia); *clinical course* (slowly progressive in giant cell tumors with tendency to break through the periosteum and to invade the surrounding soft tissue, frequent recurrence following operation; relatively benign course in osteitis fibrosa, especially for the cystic form, some cases of spontaneous healing and as a rule good response to surgery) are factors which make it doubtful whether localized osteitis fibrosa and giant cell tumor represent only different manifestations of the same nosologic entity.

As far as the treatment is concerned, careful and thorough curettage in localized lesions followed by implantation of a good number of bone chips seems to be the method of choice. Roentgen treatment may be of advantage but our experience is too limited to permit a more definite statement. In more extensive giant cell tumors with soft tissue involvement, amputation has to be considered as the quickest way to permanent cure, if roentgen treatment or less radical surgical means fails to improve the condition.

MELANOMA OF THE LUMBAR REGION WITHOUT APPARENT SKIN MANIFESTATION

CASE REPORT

By S. K. LIVINGSTON, M.D., F.A.C.S.
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MALIGNANT melanomas (neuro-epithelioma, melanin producing) embrace tumors of widely diversified histologic structure and origin.[3] They may occur in situations other than the skin. Cases

FIG. 1. Roentgenograms of spine show nearly complete destruction of the spinous and transverse processes and laminae of the second and third lumbar vertebrae, with partial destruction of the body of the third lumbar vertebra and beginning destruction of the spinous processes of the fourth and fifth lumbar vertebrae.

have been reported in various viscera in the absence of any apparent abnormality of the skin.

Malignant cells of a melanoma may spread by (1) direct extension (as is probable in the case reported); (2) lymphatics to regional lymph glands, and (3) blood stream with generalization of the disease.

Geschickter and Copeland[4] cite a few cases in which metastasis to bone occurred. In their laboratory 1.07 per cent, or 3 in 169 cases, showed secondary bone deposits. In these cases the location of the deposit was at the site of the nutrient artery.

The following report is presented as an unusual manifestation of melanoma developing in the left lumbar region without apparent abnormality of the skin, and involving, by direct extension with complete destruction, the laminae, transverse and spinous processes of the second and third, with partial destruction of the body of the third and beginning destruction in the body of the fourth and fifth lumbar vertebrae.

CASE REPORT

R.M.J. (No. 60391), a white male, aged thirty-five, occupation lumberman, was admitted March 28, 1935, for treatment of sarcoma of the spine.

Chief Complaint. Pain in back, right hip and crippled right leg.

Family History. Essentially negative.

Past History. Pneumonia at the age of six, malaria at the age of eight and seventeen. Appendectomy in 1920. No history of moles.

Present Illness. In August, 1934, stiffness in the lower back was first noticed, which gradually increased with the development of a dull pain. Roentgenograms taken in September, 1934, were negative. About March 1, 1935, symptoms of stiffness and pain became much worse and roentgenograms taken elsewhere at that time revealed partial destruction of the body of the third lumbar vertebra.

Physical examination reveals a well developed and nourished male who is not acutely ill and whose general physical examination is essentially negative. Standing is impossible because of pain in the back. Palpation of the spine reveals no gibbus but there is extreme tenderness over the spinous processes of the second to fifth lumbar vertebrae. There is a large, firm, palp-

able mass in the left lumbar region. There is a full range of motion in the lower extremities. Superficial and deep reflexes of the lower extremities are increased and there is some loss of sphincter control.

The neurological examination reveals no evidence of cord involvement.

The tumor, which on deep palpation is somewhat fluctuant, was aspirated and 8 c.c. of old, bloody fluid was removed.

Examination of the chest was negative.

Roentgen examination shows nearly complete destruction of the laminae of second and third lumbar vertebrae on the left and the spinous processes, partial destruction of the body of the third lumbar vertebra with beginning destruction in the fourth and fifth spinous processes, suggestive of malignancy.

Gastrointestinal series and roentgenograms of the skull and long bones show no anatomical or pathological variation from the normal.

Laboratory Examination. Urine, negative; Bence-Jones bodies not found. Wassermann and Kahn reactions, negative. White blood cells, 4,400,000; white blood cells, 12,000; hemoglobin, 80 per cent; polymorphonuclear leukocytes, 75 per cent; lymphocytes, 18 per cent; monocytes, 7 per cent. Blood calcium, 1.9 mg. per 100 c.c. Spinal fluid: pressure, 40 mm. Hg; colorless; globulin, 1 plus; Wassermann reaction, negative; colloidal gold, 0012221100; protein, 100 mg. per 100 c.c.; sugar, 40 mg. per 100 c.c. Aspirated fluid shows many pus cells, few gram-negative bacilli. No tubercle bacilli found on direct smear; guinea pig inoculation also negative for tubercle bacilli. Centrifugal fluid shows small areas of brownish pigment with few cells which cannot be identified.

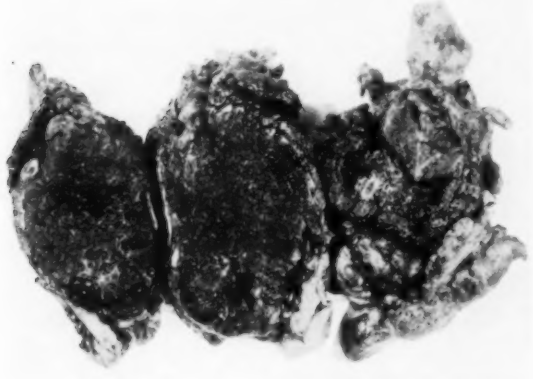

Fig. 2. Gross specimen of tumor removed surgically showing many islands of black pigment.

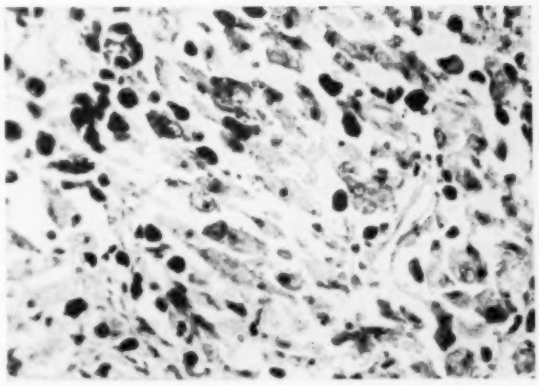

Fig. 3. Section taken from the tumor showing cells containing brown-blackish pigment in some areas. These are arranged around the blood vessels and there is a tendency to small island grouping. The stroma is moderate in amount. Degeneration is noted in places. Diagnosis: Neuro-epithelioma, melanin producing.

Tumor removed May 15, 1935. Section shows tumor cells containing brown-blackish pigment. In some areas there are arranged around the blood vessels small island groupings. Stroma is moderate in amount. Degeneration is noted in places.

Diagnosis. Neuro-epithelioma, melanin producing.

Postoperative Treatment. Wound healed satisfactorily. Modified Mayo-Fuller brace provided and a total of 1,847 r deep roentgen therapy was given. Sphincter control has returned and the patient is now ambulant. Discharged June 17, 1935, improved.

SUMMARY

1. An unusual manifestation of a neuro-epithelioma, melanin producing, in the lumbar region without apparent abnormality of the skin, is presented.

2. Skeletal metastasis by direct extension to the spinous and transverse processes and laminae of the second and third lumbar vertebrae with complete destruction was noted. There was also partial destruction of the body of the third lumbar vertebra

and beginning destruction of the spinous processes of the fourth and fifth lumbar vertebrae as noted in the roentgen examination.

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Vol. 37, No. 1

THE TREATMENT OF PROSTATIC CARCINOMA*

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IT IS unnecessary for me to dwell upon the difficulties which confront us in trying to cure patients with prostatic carcinoma. There is little disposition on the part of most urologists even to attempt to indicate a percentage basis of curability. I believe, however, that some of these difficulties will disappear if one carefully analyzes the reasons therefor.

Briefly the prostate is a semi-internal organ. It can be palpated rectally. It can be seen in outline by means of a somewhat painful maneuver, cystoscopy. Specimens of the gland for pathological examination have been difficult to obtain. The prostate is situated at what might be termed one of the important junctions, the vesico-urethral. If that junction is disturbed, confusion arises in a vital body organ, the kidneys. The prostate, particularly when enlarged, is a gland of several planes which have to be taken into consideration when either operation or irradiation is considered. There are differences of opinion in regard to the part or parts of the prostate in which the carcinoma originates. It would be presumptuous for me to suggest that I am able to give the complete answer to all of these problems. All that I can hope to do is to offer some suggestions.

These suggestions group themselves under the following heads:

1. Points of origin of the carcinoma.
2. Diagnosis by aspiration biopsy.
3. Pathological anatomy influencing radiation.

1. *The Point of Origin of Prostatic Carcinoma.* Determination of the place of origin of prostatic carcinoma is vitally important because we have the greatest chance of cure in the small carcinomas which have not extended far from the original focus. Fixing the point of origin may be done in several ways. First, by the examination of prostates of patients who have come to routine autopsy and in whom no diagnosis of prostatic cancer had been primarily made—obviously very small prostatic cancers. Young,[1] who first suggested that the majority of prostatic cancers originate in the posterior prostatic lobe, bases his belief on such examinations. He cites 2 cases in which the prostate appeared non-malignant at operation and in which on pathological examination there was carcinoma of the posterior lobe alone. The carcinoma was too small to diagnose preoperatively. In the same article Young reports several cases in which the diagnosis of carcinoma was made, preoperatively, and in which the posterior lobe was apparently the only lobe involved. He goes on to say, "The outstanding fact is that carcinoma beginning in the posterior portion of the prostate and from there involving the lateral lobes usually involves the tissue behind the trigone and around the seminal vesicles and vasa deferentia without passing into the urethral or vesical lumen."

Still dealing with small undiagnosed carcinomas which were obtained at autopsy Moore[2] summarizes the results of his examination of 52 cases.

In 52 cases the lesions were small and an accurate determination of the point of origin could be determined. 73.5 percent arose in the posterior lobe, that portion of the gland posterior and postero-lateral to the fan-shaped ducts which enter the sides of the urethra. Six or 8.8 percent arose in the lateral lobes, that portion medial and anterior to these same ducts. Ten or 14.8 percent arose in the anterior lobe, that portion which drains into the an-

[1] Lewis. Practice of Surgery. Vol. 9, Chap. 21.
[2] Moore, R. A. Morphology of small prostatic carcinoma. *J. Urol.*, 1935, *33*, 224–234.

* Read at the Thirty-sixth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 24–27, 1935.

terior furrow of the urethra. None have been observed in the middle lobe, that portion between the urethra and deferential canal and cephalad to the colliculus seminalis. In 1 case there was complete carcinomatous infiltration of the middle lobe but the posterior lobe was also involved and the focus of origin could not be determined.

So much for the small, clinically undiagnosed and undiagnosable prostatic carcinoma. When the prostatic carcinoma reaches sufficient size to permit a diagnosis we are faced with another difficulty. Young, in 27 prostatic cancers which he thought small enough for radical resection, reports only one case, if I read his report correctly, in which the posterior lobe alone was carcinomatous. Randall,[3] examining still larger autopsy specimens, says:

> There is no evidence found in this series to substantiate a previous conception that is prevalent in the literature that malignancy of the prostate has its origin in the posterior lobe. In the majority of specimens the malignant tissue appears to involve both lateral lobes quite uniformly and one finds no evidence of growth beginning posteriorly and spreading therefrom or comprising normal glandular tissue as seen in benign hypertrophy.
>
> Two surgical lesions in gross pathology may be drawn from this group of 17 cases of prostatic cancer. The first is that malignant proliferation may have its origin in any portion and involve any part of the prostate gland. The older teaching of the importance of the role played by the posterior lobe is no longer acceptable.

Randall, in giving this opinion, doubtless, and probably rightly, had in mind the practical problem presented by carcinoma of the prostate large enough to be diagnosed.

On the other hand, I am convinced that prostatic carcinoma may originate in the posterior lobe, invade the perivesicle lymphatics and grow up behind the bladder and cause death without even invading the lateral or subcervical group (median) lobes.

[3] Randall, A. Surgical Pathology of Prostatic Obstructions. Williams and Wilkins, Baltimore, 1931, p. 208 and 214.

The following case demonstrates this:

T. F. (Memorial Hospital No. 33220), admitted April 14, 1924. At that time his prostate on direct examination was large, irregular and very hard, more indurated on the left side extending just over the median line. His residual urine was 375 c.c. Cystoscopy showed both lateral and median lobes enlarged moderately, but smooth in outline. There appeared to be an adenomatous enlargement of these lobes. Suprapubic prostatectomy was done. The pathological examination of the removed portion showed no carcinoma, yet carcinoma of the posterior lobe was diagnosed and treated once. He did not return for treatment and died in September, 1926, from the carcinoma.

From all these data we may deduce the following facts: Carcinoma in the great majority of cases originates in the posterior lobe. In most cases there is one focus of origin. If the carcinoma is of sufficient size to be diagnosed clinically, the posterior lobe is almost certain to be involved.

The lateral and subcervical lobes may be involved secondarily or more rarely they may never be involved.

There may be multiple foci of origin of the carcinoma; that is, in the posterior, lateral, subcervical or anterior lobes.

One other important fact concerning the extension of the carcinoma has been brought out by Moore, namely, "Perineal lymphatic invasion in the capsule of the prostate is one of the earliest changes in carcinoma but invasion of vesicles and distant lymphatic invasion is a late manifestation."

2. *Diagnosis by Aspiration Biopsy.* It is fortunate that when carcinoma occurs in the prostate it is the posterior lobe which is so often affected as this is not only the easiest lobe to palpate rectally but is in the most convenient place for aspiration biopsy. Aspiration biopsy of the lateral lobes is more difficult while an aspiration biopsy of the subcervical group is very difficult—difficult in the sense of being sure of the location of the point of the aspiration needle. Prostatic tissue can be obtained in about 80 per cent of cases in which aspira-

tion biopsy is attempted. The technique has been too often described to make it necessary for me to give such details here.

The pathologist who examines the slides must be sufficiently well trained to make the diagnosis.

The proper use of aspiration biopsy in early suspected cases of prostatic carcinoma is one of the important factors in solving the question of the control of these cases.

3. *The Anatomy of the Prostate.* The normal prostate has been described as a cone with its base at the bladder end and its apex pointing downward. The apex is at the junction of the membranous and prostatic urethra. The only portion which shows within the bladder is the somewhat flat base of the cone. The entire prostate is below the bladder.

Because prostatic carcinoma starts so often in the posterior lobe we are particularly interested in its anatomy.

Lowsley's[4] classic description of this lobe in the prostate removed from a seventeen year old boy is as follows:

> This lobe is separated from the lateral lobes by a partition, which is quite thick and is composed of white elastic and fibrous connective tissue, with occasional smooth muscle fibers interspersed. There are some places where this tissue is thin, so that the separation between lateral and posterior lobes is not particularly marked. The amount of prostatic tissue in the posterior lobe varies according to the location in the cross section at which it is observed. This lobe occurs in the shape of a wedge with its base at the apex of the prostate, its apex being found posteriorly from the point where the ejaculatory ducts begin their oblique passage through the prostate. The posterior lobe, it is therefore seen, is the only portion of the gland which is palpated per rectum, except in a very few instances in which the lateral lobes are greatly enlarged and may be felt on the borders of the gland.

I have had cut for pathological examination complete sections of normal prostates obtained at autopsy. These show the position of the posterior lobe and how completely it is *within the urethra.* Appreciation of this fact is important when irradiation is contemplated.

[4] Lowsley. *Tr. Am. Urol. Ass.*, 1915.

TREATMENT OF PROSTATIC CARCINOMA

Operative. Young who originated the operation for the radical removal of prostatic carcinoma shows but comparatively few cases operated upon, when one considers the enormous amount of material at his disposal. In the cases operated upon he shows some brilliant five year cures. No other urologist that I know of shows even comparable results.

Considering the great difficulties of operative removal of the carcinomatous prostate and the comparatively few cures, we have had to turn to some other method and that has been irradiation.

The difficulties involved in proper irradiation of the carcinomatous prostate have been and still are very great. Obviously that is so or there would be more five year controls reported.

I believe it is generally conceded that external irradiation by 200 kv. is not enough to control the great majority of prostatic carcinomas. Even the best method, that of Coutard, giving 300 r daily to 4 or 5 portals of entry so that each portal receives between 1,500 and 1,800 r is not sufficient to control the disease. External irradiation must be supplanted by some form of irradiation within the prostate itself. And this is not as simple as it sounds.

The suprapubic implantation into the prostate of radon seeds is comparatively simple as far as the implantation of the lateral and subtrigonal lobes is concerned. But if we turn to the section of the prostate showing the posterior lobe, the lobe that is wholly subvesical we appreciate how difficult any implantation of this lobe from the vesical side may be. The posterior lobe is almost always involved in the growth, and suprapubic implantation must be supplemented by perineal treatment of this lobe.

If we are reasonably sure that there is no involvement of the lateral and subtrigonal

lobes, if the cancer is confined to the posterior lobe, then the ideal method of irradiation is through the perineum by means of radon needles which are inserted in much the same way as an aspiration biopsy is obtained. The tissue of the posterior lobe, the periprostatic tissue and the perilymphatic invasion around the seminal vesicles can be easily reached by this method. This perineal irradiation must be repeated at intervals until the prostate is sclerosed. Radon seeds may be implanted perineally but they cannot be as accurately placed as the removable needles.

The aspiration biopsy, cystoscopy, examination of sections removed from the bladder neck if there is urinary retention, a knowledge of the pathology of prostatic carcinoma, all these ought to indicate exactly what portions of the prostate are involved in the growth. Intraprostatic irradiation as above outlined is then carried out. Such irradiation is always supplanted by external irradiation.

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SARCOMA OF THE SOFT TISSUE*

By IRA I. KAPLAN, B.S., M.D., *and* SIDNEY RUBENFELD, B.S., M.D.
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DURING the period from 1924 to 1934 there were admitted to the Radiation Therapy Service at Bellevue Hospital 3,750 new cases of malignant disease. Of these, 162, or 4.3 per cent, were classified as sarcomas.

Our study of these 162 cases disclosed that 78, or 48.7 per cent, clinically and pathologically were interpreted as soft tissue sarcomas. This group includes only those tumors originating in the skin, muscle or fascial structures which manifested themselves on, or eventually infiltrated through, the skin. In our present discussion we have excluded melanosarcoma of the eye, and retroperitoneal sarcomas, which, although they are soft tissue tumors, do not involve, or make their appearance in the skin. Also excluded from this group is lymphosarcoma because these tumors are classified as lymphogranulomas. Such skin sarcomas as Kaposi's, or multiple idiopathic hemorrhagic sarcomas, were also omitted, because they appear as distinct disease entities.

Analyses of large series of cases of soft tissue sarcoma appear rather infrequently in the literature. Bick[1] has reported 20 cases in one series, and 17 cases in another;[2] Schreiner and Wehr,[18,19] 23 cases; French,[10] 16 cases; Stewart and Copeland,[23] 64 cases; and Quick and Cutler,[16] 72 cases. In the series herewith presented there are, as already mentioned, 78 cases of soft tissue sarcoma from the lesions situated in various parts of the body.

To present comparative figures for statistical analysis of this type of tumor is somewhat difficult because (*a*) "soft tissue" is not always interpreted alike by all observers; and (*b*) the term "neurogenic sarcoma" has of late come to be applied to those types which formerly were called fibrosarcoma and spindle cell sarcoma.

As to the etiology of these tumors, it appears that sarcoma rather than carcinoma has been associated most often with a traumatic origin. Whether or not trauma is an important etiological factor in soft tissue sarcoma is, as yet, a moot question. Löwenthal,[12] in 1895, listed 358 out of 800 tumors that were preceded by acute trauma. Coley[6] has stressed its importance for many years and as early as 1897 he recorded 46 out of 170 cases of sarcoma with an antecedent traumatic history. In 1910 he[7] cited 225 instances in a series of 970 sarcomas; and in bone sarcoma the causal relationship presented an even higher frequency, i.e., 50 per cent. Coley and Higinbotham[9] did not hesitate to connect the factor of trauma with neoplastic growth by the microorganism theory, and stated, "We may assume that malignant tumors in man are due to a micro-organism that is latent in the circulation, and which gives rise to symptoms of malignancy only after the normal resistance of the cells is broken down in some instances by local trauma. The micro-organism thus finds a suitable soil in the damaged cell, forming a symbiosis with the cell and causing a proliferation and multiplication resulting in a malignant tumor." From our clinical experience, we cannot give assent to such a hypothesis. However, the following case in our series would seem to lend some support to such a speculative theory:

CASE 56. F. K., aged fifty-two, tailor, Austrian, admitted May, 1931. Twelve years before admission he noticed a small pimple on the left side of his back which slowly increased in size until it attained a dimension of about 1 inch in diameter. It remained this size until several months prior to admission, when it began to grow more rapidly without producing pain or bleeding.

Concurrently, twelve years before admis-

* From the Radiation Therapy Service, Bellevue Hospital, New York City, Ira I. Kaplan, M.D., Director. Read at the Thirty-sixth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 24–27, 1935.

sion, he developed an egg-sized lump in the lateral field of the left supraclavicular area. This tumor mass grew very slowly, produced no symptoms, always remained mobile, and not until several months before admission did it show any evidences of expansion. By that time, it had attained the size of an orange and was surrounded by two smaller nodules of the same nature.

A biopsy of the lesion on the back showed it to be basal cell epithelioma, and that of the nodule on the neck a spindle cell sarcoma.

Evidently then, after remaining latent twelve years, and without antecedent trauma, both tumors began to grow rapidly. Under Coley's theory, we may adopt the hypothesis that this long-dormant microorganism was stimulated to activity by an unknown source giving rise to two distinct type tumors in different sites.

Knox,[11] in 1929, definitely ruled out trauma as an etiological source of tumors. She placed considerable weight upon the inability of a single trauma to produce tumors experimentally. Lubarsch[13] and Ribbert[17] were both unsuccessful in producing neoplasia, whether by simply traumatizing tissue or by injecting traumatized tissue in animals. Knox reasoned that as a result of a single trauma scars persist as demonstrable lesions because of the presence of foreign material implanted in the beginning, or possibly because of an infection. Therefore, rather than acute single trauma, the important factor is that of chronic irritation. Such a sequence would obtain support in the report of Bloodgood,[4] who found that of 48 sarcomas of the skin, the neoplasms developed in a nevus 8 times, in a scar 20 times, and in a fibroma, 9 times. Recently, Simenstad[21] cited the case of a sarcoma of the hand which developed following two years of various forms of therapy, i.e., frequent incisions, hot applications, and blisterings, all of which suggest a chronic irritative rather than a traumatic etiology.

In evaluating the rôle of trauma in the production of the stromal tumors in this series, we adopted as our criteria the following qualifications, suggested by Segond[20] in 1907 and accepted by Ewing:[9]

I. The authenticity and sufficient severity of the trauma.
II. The previous integrity of the wounded part.
III. The identity of the injured area with the site of the subsequent tumor.
IV. The tumor must be shown to be the type which could result from trauma.
V. A proper time interval must elapse following injury.

In our series, 10 patients, or 13.1 per cent, gave an antecedent history of trauma. Quick and Cutler reported 19.3 per cent in their series, and Stewart and Copeland, 12.5 per cent. Most cases in our series fitted into only the first four of the five criteria listed above. The time interval proved too variable a factor, and one capable of permitting too much of the personal equation.

CASE 27. M. O., aged forty-nine, female, single, American, admitted November, 1931, with a tumor on the mesial aspect of the right ankle. In 1911, twenty years earlier, she had been struck over this area with a 50 pound weight. There was the usual sequence of contusion followed by a complete cure. Fourteen years later, in 1925, she noticed a nodule one-quarter inch in diameter over the same site. It was excised but recurred, and was again excised within the same year (1925). The lesion was symptomless until May, 1930 (five years later), when another nodule reappeared over the same location. Excision this time was followed by slow healing, and ultimately, by an ulcerative, exudative, malignant lesion.

CASE 10. J. B., aged thirty-four, laborer, Polish, admitted in 1926 with a bleeding mass over the left arm. He had had an egg-sized nodule on the arm for thirty years without ever experiencing any symptoms; but two days prior to admission he was struck over this area, with consequent bleeding, pain, and sudden, diffuse enlargement of the mass, which on biopsy, proved to be malignant (Fig. 4).

In reference to these 2 cases, what may be stated as the proper time interval? . . . twenty years, as in the one instance, or two days, as in the other? Obviously, we cannot possibly determine the bridging symptoms

and say that the latent period has such a wide range.

In our opinion, based entirely upon clinical factors, the patient, the tumor, and the traumatic effect must, in every instance, be individualized, since the sequence is apparent in some cases and dubious in others.

Age. As to age, 44 cases, or 56 per cent, appeared during the third to the fifth decade inclusive, an incidence which, more or less, runs parallel to the general occurrence of malignancies. About one-fourth (22.9 per cent) of the patients were less than thirty years of age, a relatively high incidence in the young as contrasted with the age incidence in other types of malignancy. The youngest case was two, and the oldest seventy-five years old. In 2 instances of sarcoma in the scapular region, one was in a two year old child, the other in a seventy-two year old man. This illustrates the extreme variability of age distribution.

Sex. Fifty-five patients, or 70 per cent, were males; and 23, or 30 per cent, were females, a ratio of 2.3 to 1. This relatively high incidence in males is greater than that reported by Pack and Lefevre[15] of 61 per cent males to 39 per cent females; or Stewart and Copeland's report of their series with a ratio of 1.3 to 1. We can offer no satisfactory explanation for this obvious, but inconsequential, sex ratio discrepancy. In general, however, most statistics agree as to the predominance in males.

Location. According to most reports, sarcomas of the soft tissue are present most frequently on the extremities, with the lower limb the usual site of predilection. Thus, Bland-Sutton[3] states that "sarcoma of the nerves shows a distinct preponderance for the lower limbs." Quick and Cutler reported 44 per cent of such tumors occurring in the lower, and 20 per cent in the upper extremity. In his recent analysis of cases of muscle sarcoma, Bick[1] cited 16 of 20 cases as appearing in the thigh, abdominal wall, and forearm. A later report by this same author[2] cited 17 cases of fibrosarcoma in the extremities. In our cases, the distribution was as follows (Table I).

Table I

Location of Tumors

Area	Right	Left	Total
Inguinal region	3	3	6
Arm	1	4	5
Forearm	2	0	2
Scapular region	0	3	3
Hand	2	0	2
Foot	9	1	10
Leg	3	6	9
Thigh	6	12	18
Neck	2	2	4
Chest	2	2	4
Abdomen			3
Back	1	3	4
Chin			1
Ear	1		1
Nose			1
Scalp			2
Generalized			2
Ischiorectal fossa			1
			78

Thus, it is seen that in 37 instances, or 41 per cent, the tumors manifested themselves on the lower extremities; and the thigh is, by far, the most frequent site.

The side of the body on which the tumors appear is of little significance since such wide discrepancies exist. For example, the foot presented 9 tumors on the right side and one on the left side; the thigh had 12 tumors on the left, and 6 on the right.

Interval before Admission. It becomes increasingly evident from clinical experience that these sarcomas are not as rapidly fatal as carcinomas. Obviously, the epithelial neoplasms occur in more indispensable points . . . in parenchymatous organs where proper functioning is essential. However, from the therapeutic viewpoint, we received most cases long after neoplastic development had taken place. Table II indicates the time interval between the appearance of the tumor and presentation of the patient at our clinic. In most instances the gross appearance of the mass was the initial clinical symptom, with pain, bleeding, and disability as subsequent complaints.

TABLE II

INTERVAL BEFORE ADMISSION

	Inguinal Region	Arm	Forearm	Scapula	Hand	Foot	Leg	Thigh	Neck	Chest and Abdomen	Back	Chin	Ear	Scalp	General	Ischiorectal Fossa	Total
0–1 mo.		1	1					1									3
2–6 mo.	3	1		2	2	2		3	3	3		1		1	1		22
7–12 mo.						2	1	2			1						6
1–2 yr.	1	1	1			1	2	4		1			1				12
2–3 yr.				1		3	2	4		1				1			12
3–4 yr.	1					1	2	3		1	3						11
6–7 yr.						1		1								1	3
7–8 yr.		1															1
12 yr.									1								1
20 yr.										1							1

It is astonishing to note that 35 patients (44 per cent) came to the clinic one to four years after the tumor had manifested itself, and only 25 patients (32 per cent) sought aid early—within six months. This, we believe, can be essentially interpreted as clinical evidence of the slow growth of soft tissue sarcomas. Three cases had tumor masses for two years or more without evidence of rapid growth; and in 2 of the 3 cases cosmetic defects impelled the patients to seek medical attention.

Pathology. These tumors were often large, deep-seated, fixed masses, of varying consistency, depending upon the vascularity, and the underlying stroma. Tenderness was usually absent, and since some observers may interpret these tumors as

TABLE III

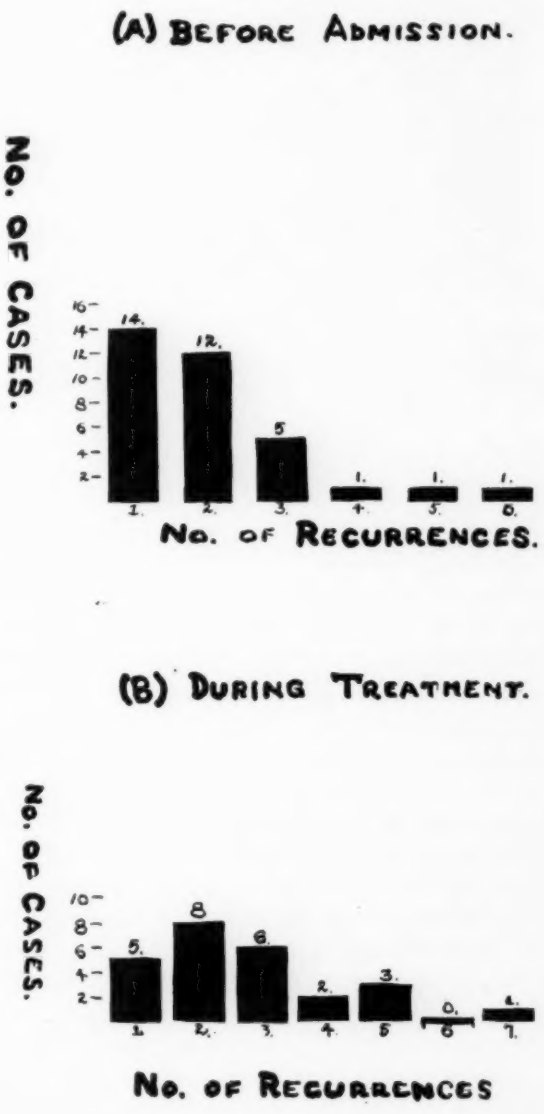
HISTOLOGICAL TYPE OF TUMORS

	Inguinal Region	Arm	Forearm	Scapular Region	Hand	Foot	Leg	Thigh	Neck	Chest and Abdomen	Back	Chin	Ear	Nose	Scalp	General	Ischiorectal Fossa	Total
Melanosarcoma	1	1		1	1	4	2	4		2	1					2		19
Spindle cell sarcoma	1	2				3	4	6	1	1	1		1		2			22
Giant and spindle cell sarcoma				1		1	1	3	1									7
Myxosarcoma	1	1						1			2							5
Round cell sarcoma	2	1							1					1				5
Perithelioma							1			1								2
Fibrosarcoma			1	1	1	2	1	4	1	3		1					1	16
Unclassified sarcoma	1		1															2
																		78

arising from neurogenic structures, this negative clinical finding assumes a significant note. Fibrosarcoma presents, invariably, a very hard consistency; myxosarcoma, on the other hand, will yield a soft, fluctuant, plum-like sensation on palpation. These tumors, arising in the deeper layers of the skin and fascia about the muscles, are, as a rule, poorly circumscribed, although some small tumors indicate clear attempts at encapsulation. However, when a tumor attains bulkiness, it grows expansively, often with a well-defined blood supply, and whatever capsule existed is effaced by the progressive growth. The growing periphery penetrates into the neighboring connective tissue framework to form adhesive bands. Such expansive penetration may be very cellular, forming thick clumps of cell masses extending in all directions and as far down as the bone, making complete excision impossible. Recurrences characterize these sarcomatous neoplasms. From our series of 78 cases we found a relatively high rate of recurrences, as indicated in Chart 1.

Case 70. This case presents a notable history to exemplify this characteristic. N. L., male, aged thirty-two, clerk, Austrian, admitted in October, 1931. In 1928 he had a tumor removed from the left lumbar region, but one year later it began to reappear; and in 1931, when it had attained the size of a large grapefruit, it was again excised. Soon after he presented himself to us and roentgen therapy was administered to the involved areas. In February, 1932, two small nodular recurrences appeared and these were treated with radon seeds, a dose of 600 mc-hr. being given. The lesion healed well. In October, 1932, another large nodule appeared, which was successfully irradiated with 6,000 mg-hr. of platinum-filtered radium element needles. By January, 1933, a non-ulcerated recurrence was evident and this was resected with the endotherm. The wound healed well, and 50,000 mg-hr. of gamma radiation by means of the 5 gram pack was employed in December, 1933, to treat a recurrence in the resected area. When this failed, in January, 1934, the lesion was again excised with the endotherm. By October, 1934, another tumor 5 cm. in diameter appeared along with an enlarged, fluctuant, inguinal node on the same side as the lumbar tumor, and in November the mass and the node were resected with the scalpel. Both operative sites healed without complications, and the patient is at present being kept under observation. This tumor recurred eight times.

Chart I



Microscopic Pathology. Ewing[8] states that it is desirable to avoid terms referring solely to the morphology of cells, and that such descriptive terminology as spindle, round, and giant cell sarcoma convey little

FIG. 1. Case 21 (R.B.). A very malignant melanosarcoma arising from the web spaces of the foot of a woman aged seventy-four. Patient was dead within four months.

information as to the origin of the tumor. He observes, furthermore, that cell arrangement often reflects the structure of the mother tissue, but that in very cellular growths, this lineage is lost. We would like to establish this correlation and stamp microscopic pictures to characterize the tissue from which a neoplasm arose; but we were unable to arrive at sufficiently satisfactory conclusions. Hence we cannot agree with Stewart and Copeland that neurosarcomas constitute the bulk of fibrosarcomas of the soft parts.

Table III indicates the histological type of the tumors occurring in the various anatomical sites. Most of these slides were read and interpreted by Dr. Douglas Symmers, Pathologist of Bellevue Hospital, to whom we are indebted for the pathological reports on the cases herein presented. The section of one of our cases, a tumor of the thigh, showed numerous strands of spindle cells arranged in whorl fashion, lying in a loose stromal network of moderate vascularity. Such a section suggests its neurogenic origin, but in the absence of nerve structures, or nerve producing structures, we are not willing to employ that terminology. We believe that wherever cellular tumors are found, final judgment as to their nature should be left to the gross dissection, and even this procedure may not yield conclusive evidence.

The high frequency of spindle cell sarcomas and fibrosarcomas (48.7 per cent), should be noted. Many of these may be listed by others as neurogenic sarcoma. The section taken from Case 52, that of a tumor of the thigh, was interpreted as spindle cell sarcoma, wherein large spindle and polygonal cells with pale staining vesicular nuclei were found within an abundant fibrillar stromal network. Such a picture would coincide with the Grade II picture outlined by Quick and Cutler.

The histopathology of melanoma yielded little difficulty as far as diagnosis was concerned. It is not our aim to advance any factors in the solution of the origin or pathogenesis of these tumors; but, since all were on the skin, we assume that their origin was from the chromatophores of that tissue. The uncertainty of the origin of these tumors is elaborately discussed by

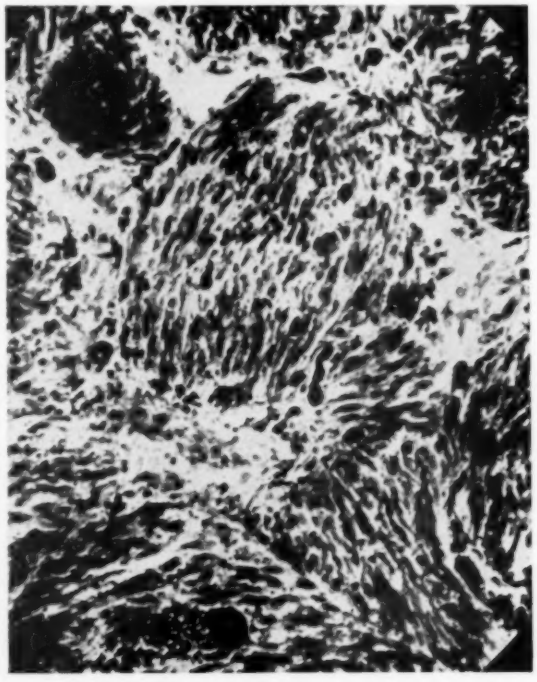


FIG. 2. Case 21. Depicting the histology of the tumor shown in Figure 1. The islands of cells have an overabundant deposit of melanin which consumes almost the entire cell outline.

Ewing, who presents the controversial opinions of many authors. Figure 1 shows a melanotic tumor over the plantar surface of the foot in a woman aged seventy-four. The microscopic picture (Fig. 2) reveals islands of compact, deeply pigmented spindle cells surrounded by areas of loosely woven, non-melanotic cells. The tumor was very resistant to radium therapy, never exhibited a primary response, metastasized early, and proved fatal. In another case of melanotic tumor the pigmented cells were less frequent, large, and oval shaped. Although eventually fatal, this mass showed an encouraging response after treatment, and remained healed for six months. An entirely different cell structure was shown in Case 36 in that the non-pigmented cells were large, round and pale staining, and the specially notable feature was the appearance of the melanin deposits entirely within the circumference of the nuclear membrane, yielding the impression as of some phagocytic property. Indeed, this patient was alive and well three years after treatment.

In this series, 5 tumors were interpreted as round cell sarcoma. Many writers have questioned the derivation of these tumors from true fibroblastic connective tissue, and call them lymphosarcoma; others emphasize the need to search for identifying arrangement, such as spindle or giant cells.

Difficulty is also encountered when one employs the term "mixed cell sarcoma." However, in sections where cells of many forms and sizes occur, and where nuclear modifications are present, this term is resorted to for want of something better. Giant cells with several hyperchromatic nuclei interspaced with pale-staining spindle cells both yield a picture ably described as "giant and spindle cell sarcoma."

We cannot definitely state whether the so-called perithelioma or perithelial sarcoma is derived from the adventitial cells of the vessel wall; its microscopic appearance, however, is so characteristic that its designation is hardly ever doubted. The appearance of perithelioma is supposedly

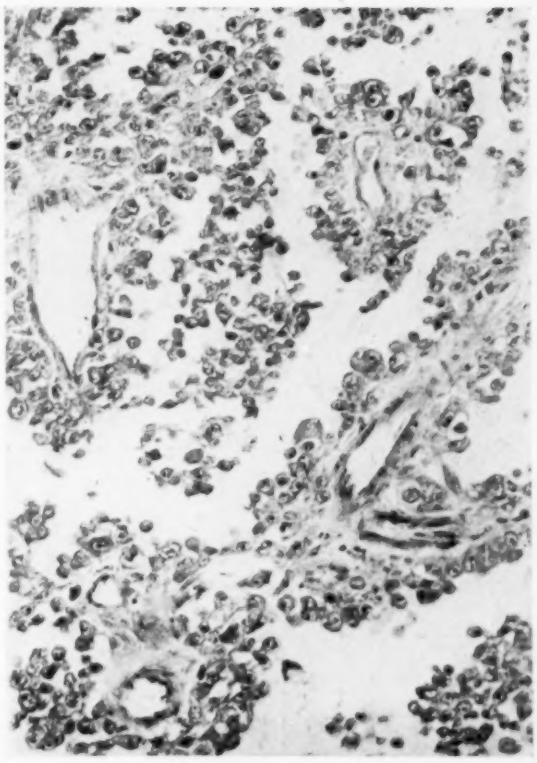


Fig. 3. Case 29 (F.A.). A perithelioma of the leg. The very characteristic units of a central blood vessel and radiating large cells are clearly visualized.

the result of the persistence of the cells intimately surrounding the vessel wall, while those more distant are dissolved by necrotic changes. Figure 3 shows a typical perithelioma; surrounding a small blood vessel are one or more layers of large, pale-staining cells which radiate from the vessel wall, and at varying distances from the vessel is a clear space, probably from lack of nutrition. In this fashion, units of (1) a vessel and (2) radiating cells are created. Such a histological association of blood vessel and tumor cells obviously suggests early blood stream invasion, which was, indeed, the course in our cases.

Treatment. All tumors in this series were surgically removed, wholly or partially, at some time during the period of observation. The form and mode of application of radium and roentgen therapy were so varied and individualized that it is impossible to chart the methods employed.

In our attempt to control recurrences, these tumors proved sufficiently baffling to evoke all the means at our disposal. Nevertheless, whenever a mass was irradiated, the neighboring lymph drainage area generally also received proper irradiation. All patients had roentgenograms of the lungs taken at frequent intervals to detect early evidences of lung metastases. The area from which a mass was excised received either roentgen or radium therapy, the method and dosage depending wholly upon the location, site, and condition of the patient. Irradiation with high voltage roentgen rays was chosen for a large tumor bed; radium in the form of needles, seeds, or on molds was used for smaller tumors. For tumor removal, whenever possible the electrocautery was employed. Small nodular recurrences usually were immediately implanted with radium applicators. Amputation of a limb was carried out in several instances, although generally this advice was thrust aside by the patient with "I would rather be dead," for such a procedure to a man of active working age often proved a difficult problem. More recently we have been utilizing the massive 5 gram radium pack, administering large destructive doses. Numerous patients were *treated merely for palliation*. Advanced tumor masses were hardly expected to yield any favorable results in spite of the most radical procedures. In no one instance can we cite the ideal method, and usually the tumor and the patient must be considered individually. Often, in the single case, we resorted to every possibility in an attempt to arrest the growth.

TABLE IV

RESULTS OF TREATMENT

	Dead												Living												No Return
	After Admission					After Onset							After Admission							After Onset								
Years	1	2	3	4	5	1	2	3	4	5	6	7	8	1	2	3	4	6	7	8	1	2	3	4	5	6	7	8	13	20	
Melanosarcoma	6	4				2		2	3	1				2		1							1		1		1				6
Spindle cell sarcoma	9		1			3	3	4						2	1		2	1				1	1	1	2						6
Fibrosarcoma	1					1								6	3	1			1		1	2	2	2		1			1	1	4
Round cell sarcoma	2						1							1			1										1				1
Mixed cell sarcoma	3	1					1	2						1							1										2
Myxosarcoma				1									1	2		2							1	1		1					
Perithelioma	1													1																	
Unclassified																															2

Table IV. Despite strenuous efforts at a careful follow-up plan, we always encounter the greatest difficulty in securing the return of patients. This is because of the distressingly poor economic condition of our charity patients at Bellevue Hospital, to whom moving from place to place is a common practice. We shall therefore not attempt to arrive at

a three or five year cure average, especially since many patients attended the clinic for varying periods of from one to three years, and never reappeared after the last observation when they were in good health.

Of the 19 patients with melanosarcoma, 6 failed to return; and of the remaining 13 cases, 10, or 76.9 per cent, were dead at the end of two years. Three patients were living; 2 at the end of the first year, and one at the end of three years.

The series of 22 patients with spindle cell sarcoma is, actually, limited to 16, because of the lack of report on 6. At the end of the first year after admission, 9, or 56.2 per cent, were dead; 6 patients were doing well, 2 at the end of four years, and one at the end of six years.

The theory that fibrosarcoma is a slow-growing tumor is well substantiated in this series, since of the 12 cases for consideration, only one died within the first year after admission, and 11 patients were alive and well from one to seven years after admission to the clinic. It is interesting to note that 2 patients in this long-lived series presented an antecedent history of thirteen years in the one instance, and twenty years in the other.

Fig. 4. Case 10. Spindle cell sarcoma of the arm. The patient was offered amputation, but refused. Instead, the lesion was treated by endothermic resection and radium therapy.

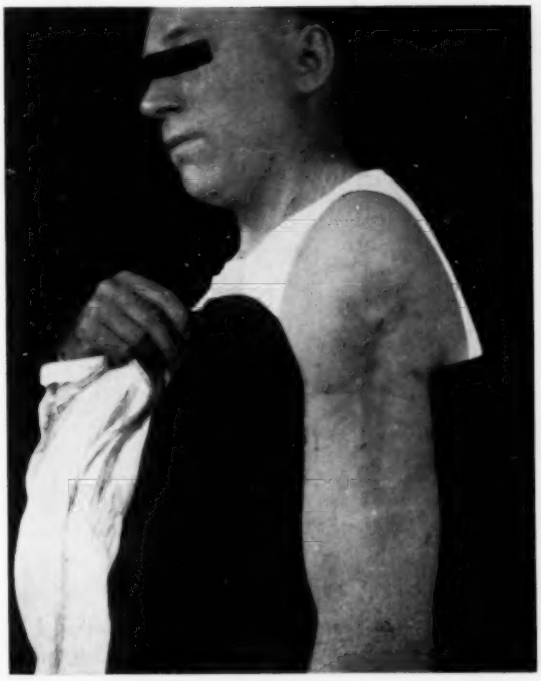


Fig. 5. Case 10. Same case as in Figure 4 showing the healed lesion after endothermic resection and radium therapy. Patient is well after six years.

The number of patients in each of the groups with mixed and round cell sarcoma are too few to yield any definite conclusions. Yet 2 of the 4 studied cases of round cell sarcoma were dead within the first year. Of the patients with mixed cell tumors, a series of 7 was reduced to 5, since 2 were lost in the follow-up. Four of these died within two years. Such records certainly suggest the characteristic of rapid growth to be associated with mixed and round cell sarcomas.

This, however, is in direct contrast with myxosarcoma, which presented clinical manifestations of slow growth. The 5 patients have all been followed up. One died at the end of four years, or eight years after the onset of the tumor. The remaining 4 are alive from one to three years.

The 2 cases listed as having unclassified tumors never returned for observation.

Perithelial sarcoma proved to be a rap-

TABLE V
METASTASES

	Lungs	Nodes	Liver	General	Other Sites
Spindle cell sarcoma	4	1			Jaw and toe Spine and skull
Melanosarcoma	2	4	2	4	
Mixed cell sarcoma	4				
Perithelioma		1			

idly growing tumor in the 2 recorded cases. One patient died after one month, and the second presented large metastatic glandular deposits within eight months.

Amputation. In only 7 instances was amputation of a limb, or part of a limb, performed. One patient is well four years subsequent to a disarticulation at the shoulder joint; another, who had a similar procedure, could not be traced. Two patients had leg amputations; one died within one year, the other is well at the end of five months. For the relief of excruciating, intractable pain, a mid-thigh amputation was performed in another instance, but the patient succumbed two months later from pulmonary metastases. There are no follow-up records of a thumb amputation and of a toe removal. Obviously, therefore, we are not justified in drawing any conclusions as to the value of amputation, or in comparing the usual manner of amputation with the radical procedures recommended by Singleton and Hyde,[22] who removed the scapula, part of the clavicle, and muscle layers in addition to the arm, for a sarcoma of that limb. One patient (Case 9) was offered amputation and accepted. She is alive and well after four years. Case 10 (Figs. 4 and 5) was offered the same procedure, refused, and was treated with local resection and radium, and is well at the end of six years. Apparently *amputation is not the complete solution to the therapeutic problem.*

Metastases. From the occurrences of metastases, as shown in Table V, several illuminating conclusions were reached. As contrasted with myxosarcoma and fibrosarcoma, the tumors of the spindle cell, mixed cell, and melanosarcoma types are much more prone to metastasize. Spindle and mixed cell sarcomas show a definite tendency to deposit foci in the lungs; melanosarcoma, on the other hand, manifests its dissemination by a generalized involvement as well as by deposits in the draining lymph nodes. The liver was involved secondarily only where melanosarcomas disseminated. Tumors of the spindle cell variety alone yielded metastases in unusual sites; i.e., the skull in one case, the jaw and toe in another, and the dorsal vertebrae at the level of the fifth root in the last case where a total paralysis below the lesion was produced.

SUMMARY AND CONCLUSIONS

1. A series of 78 soft tissue tumors are analyzed from clinical, pathological, and therapeutic viewpoints.

2. These tumors constituted a frequency of 48.7 per cent of all sarcomas admitted to the Radiation Therapy Service at Bellevue Hospital.

3. Ten patients presented histories with authentic antecedent trauma.

4. Since the bridging symptoms and the latent period between trauma and tumor development were so varied, we are unable to state definitely that trauma plays an important rôle in producing sarcoma.

5. The tumors appeared most often during the third to the fifth decade, but 25 per cent of the cases were patients under thirty years of age.

6. Males predominated in a ratio of 2.3 to 1.

7. This type tumor was most frequently located in the extremities, especially the thighs.

Table VI

Case No.	Age	Sex	Trauma	Interval	Previous Therapy	Site	Pathology	Treatment	Results	Remarks
1 S.C.	43	M	o	8 yr.	8 yr.	Inguinal left	Melanosarcoma	S—X	No recurrence 1 mo.	Diagnosis of hernia
2 T.M.	60	F	o	6 wk.	o	Inguinal left	Round cell sarcoma	S—X	No recurrence 3 mo.	Diagnosis of hernia
3 E.G.	50	F	o	1½ yr.	o	Inguinal right	Sarcoma?	S—X	No recurrence 3 mo.	Diagnosis of hernia
4 L.A.	47	M	o	3 yr.	o	Inguinal left	Myxosarcoma	S—X	Well 3 mo.	
5 F.L.	60	M	o	2 mo.	o	Inguinal right	Round cell sarcoma	S—X	Dead 2 mo.	
6 A.Z.	24	M	o	6 mo.	o	Inguinal right	Spindle cell sarcoma	S—X	Dead 1 yr.	Metastases to lungs, jaw, and toe
7 L.W.	42	F	o	1 yr.	10 mo.	Left arm	Myxosarcoma	S–X–R	Well 3 yr.	Recurrences controlled by seeds
8 R.G.	17	F	o	1 mo.	o	Left arm	Spindle cell sarcoma	X	Dead 1 mo.	
9 F.C.	34	F	o	7 yr.	5 excisions	Right arm	Round cell sarcoma	R.-Amputation	Well 4 yr.	
10 J.B.	34	M	2 days struck		o	Left arm	Spindle cell sarcoma	X—R	Well 6 yr.	
11 F.E.	25	M	o	4 mo.	o	Left arm	Melanosarcoma	S—X	Dead 1 mo.	
12 F.G.	25	F	o	1 yr.	2 excisions	Right forearm	Fibrosarcoma	S–X–R	Well 2 yr. No recurrence	
13 S.L.	67	M	Struck by wood	2 wk.	o	Right forearm	Sarcoma?	None	No recurrence	Axillary metastases
14 G.H.	2	M	o	2 mo.	o	Left scapula	Fibrosarcoma	S–X–R	Well 6 mo.	
15 M.F.	72	M	o	6 mo.	o	Left scapula	Spindle, giant cell sarcoma	S—X	Dead 2 yr.	
16 J.S.	33	F	o	2 yr.	o	Left scapula	Melanosarcoma	S—R	Well 1 yr.	
17 L.R.	68	M	Struck by hammer	6 mo.	o	Right thumb	Melanosarcoma	Amputation	No recurrence 1 mo.	
18 M.M.	38	F	o	2 yr.	o	Right palm	Fibrosarcoma	S—X	No recurrence	
19 L.S.	30	M	o	4 mo.	o	Right foot	Spindle cell sarcoma	S—R	No recurrence 3 mo.	
20 C.V.	43	M	o	2 yr.	2 excisions	Right foot	Melanosarcoma	S—R	Dead 2 yr.	3 recurrences
21 R.B.	74	F	o	2½ yr.	o	Right foot	Melanosarcoma	X—R	Dead 4 mo.	
22 T.H.	40	M	Fissure in web	3 mo.	o	Right foot	Melanosarcoma	X—R	Dead 1 yr.	Inguinal nodes
23 F.H.	57	M	o	1 yr.	o	Right foot	Spindle cell sarcoma	X—R	Well 4 yr.	

TABLE VI (*Continued*)

Case No.	Age	Sex	Trauma	Interval	Previous Therapy	Site	Pathology	Treatment	Results	Remarks
24 M.I.	—	F	0	7 mo.	0	Left ankle	Fibrosarcoma	S—X	Well 2 mo. No recurrences	
25 T.G.	45	M	Stabbed in toe	2 yr.	0	Right foot	Spindle and giant cell sarcoma	Amputation—X	No recurrence 1 mo.	
26 J.G.	38	M	0	3 yr.	0	Right foot	Melanosarcoma	S—R	Dead 1 yr.	Lung metastases
27 M.O.	49	F	20 yr. 50 lb. weight	6 yr.	3 excisions	Right ankle	Fibrosarcoma	S—R	Well 1 yr.	Trauma
28 F.V.	43	M	0	2 yr.	2 excisions	Right foot sole	Melanosarcoma	S—R	Dead 2 yr.	3 recurrences
29 F.A.	60	M	0	—	0	Right leg	Perithelioma	Pack	Poor 8 mo.	Nodes
30 J.P.	39	F	0	4 yr.	4 excisions	Left leg	Fibrosarcoma	Pack Amputation	Well 5 mo.	Pain
31 G.G.	—	M	0	2 yr.	3 excisions	Right knee	Melanosarcoma	S–X (5)	Dead 1 yr.	Had 7 recurrences
32 F.D.	64	M	0	2½ yr.	0	Right leg	Spindle and giant cell sarcoma	X	Dead 6 mo.	Lung metastases
33 T.S.	57	M	0	1 yr.	0	Left leg	Spindle cell sarcoma	S—X	Well 4 yr.	
34 M.C.	33	F	0	8 mo.	Amputation 5 mo.	Left leg	Spindle cell sarcoma	X	No recurrence	Metastases to 5th dorsal vertebra
35 A.M.	16	M	0	4 mo.	0	Left leg	Spindle and giant cell sarcoma	S–X–R Amputation	Dead 1 yr.	Lungs
36 D.W.	23	M	0	4 yr.	3 excisions	Left leg	Melanosarcoma	S–X–R	Well 3 yr.	
37 E.C.	16	M	0	1½ yr.	Excision 1 yr.	Right thigh	Fibrosarcoma	S–X–R (4)	Well 2 yr.	
38 F.G.	50	M	0	1 yr.	Excision 1 yr.	Left leg	Spindle cell sarcoma	S—R (3)	Well 1 yr.	
39 D.F.	17	M	0	6 mo.	0	Left thigh	Spindle cell sarcoma	S—X	No recurrence	
40 M.K.	45	F	Bumped mole	7 mo.	0	Left thigh	Melanosarcoma	S—X	Poor 2 yr.	General metastases
41 B.S.	71	M	0	2 mo.	0	Right thigh	Melanospindle cell sarcoma	S—X	Dead 3 mo.	Lung metastases
42 E.S.	50	M	0	3 yr.	2 excisions	Left thigh	Spindle cell sarcoma	Pack Amputation	Dead 1 yr.	Lung metastases
43 J.R.	62	M	Broke wood 3 yr. 6 mo.	3 yr.	0	Left thigh	Fibrosarcoma	Pack	Well 6 mo.	History of trauma
44 M.M.	42	M	0	1 yr.	0	Right thigh	Spindle cell sarcoma	S—X	Dead 6 mo.	Skull metastases

Table VI (*Continued*)

Case No.	Age	Sex	Trauma	Interval	Previous Therapy	Site	Pathology	Treatment	Results	Remarks
45 S.G.	40	M	o	2 yr.	X—3 mo.	Right thigh	Myxosarcoma	S—Pack	Well 6 mo.	
46 H.T.	31	M	o	6 mo.	o	Left thigh	Melanosarcoma	S—R	Dead 5 mo.	Node and general metastases
47 M.G.	23	F	Varicose veins	2 yr.	Excision 10 mo. X—1 yr.	Left thigh	Spindle cell sarcoma	S—R (3) Pack	Well 2 yr.	
48 S.S.	63	M	o	3 yr.	o	Left thigh	Mixed cell sarcoma	S—X	Poor 2 mo.	Lung metastases
49 E.F.	27	M	o	1 yr.	o	Left thigh	Melanosarcoma	X	No recurrence	
50 M.R.	25	F	o	6 yr.	3 yr. excision	Right thigh	Fibrosarcoma	S—X	Well 1 yr.	3 mo. after pregnancy showed recurrences
51 M.M	5	M	o	3 yr. 1 mo. rapid	o	Left thigh	Mixed cell sarcoma	S—X	No recurrence	
52 R.R.	45	F	o	1 mo.	o	Left thigh	Spindle cell sarcoma	S–X–Pack	Dead 3 yr.	Lung metastases
53 I.K.	15	F	o	1½ yr.	o	Right thigh	Fibrosarcoma	S—X	Well 6 mo.	
54 J.B.	58	M	o	2 yr.	2 yr. excision by string	Left thigh	Melanosarcoma	S—X	Well 6 mo. No recurrence	Node metastases
55 P.L.	56	M	o	9 mo.	2 excisions	Left thigh	Spindle and giant cell sarcoma	S–X–R (4)	Dead 1 yr.	5 recurrences
56 F.K.	52	M	o	12 yr.	o	Left neck	Spindle cell sarcoma	S–Pack X	Well 1 yr.	
57 S.C.	75	M	o	4 mo.	o	Left neck	Mixed cell sarcoma	S—X	Well 5 mo.	
58 C.F.	33	M	o	3 mo.	o	Right neck	Fibrosarcoma	S—X	Well 2 yr.	
59 S.F.	28	M	o	3 mo.	o	Right neck	Round cell sarcoma	S—X Pack	Well 1 yr.	
60 J.S.	13	M	8 mos. Ball	6 mo.	Excision 6 wk.	Left chest	Perithelioma	S—R	Dead 1 mo.	
61 M.S.	46	M	o	20 yr.	14 yr. excision	Left chest	Fibrosarcoma	S–X–R (2)	Well 3 yr.	
62 C.S.	66	M	o	2 yr.	o	Left abdomen	Spindle cell sarcoma	S—R	Well 1 yr.	
63 A.K.	30	F	o	1 yr.	o	Left abdomen	Fibrosarcoma	S—X	Well 1 yr.	
64 H.M.	59	M	o	4 yr.	o	Right chest	Melanosarcoma	S—R	Well 1 yr.	
65 F.D.	43	M	o	6 mo.	Excised 3 mo.	Left abdomen	Fibrosarcoma	S—X	Dead 2 mo.	

TABLE VI (*Continued*)

Case No.	Age	Sex	Trauma	Interval	Previous Therapy	Site	Pathology	Treatment	Results	Remarks
66 J.B.	36	M	0	4 mo.	Excised node—2 mo.	Right chest	Melanosarcoma	X	Dead 3 mo.	Diffuse skin metastases
67 J.K.	54	M	9 mo. Fell	7 mo.	0	Right lumbar	Spindle cell sarcoma	S—X	Dead 7 mo.	
68 F.R.	47	M	0	4 yr.	2 excisions	Left lumbar	Myxosarcoma	S—X R (2)	Dead 4 yr.	Had 6 recurrences
69 L.M.	40	F	0	3 yr.	0	Left chest Back	Melanosarcoma	S–X–R	2 yr.	Metastases over body
70 N.L.	32	M	0	3 yr.	2 resections	Left lumbar	Myxosarcoma	S. (3) X. R. (7)	Well 4 yr.	
71 L.B.	30	F	0	4 mo	0	Chin	Fibrosarcoma	S—X	A mo. No recurrence	
72 M.G.	54	M	0	1 yr.	Excision 9 mo.	Right ear	Spindle cell sarcoma	S—R (2) Pack	1 yr. Poor	Lung metastases
73 M.C.	49	M	0	1 yr.	3 recurrences in 1 yr.	Nose	Round cell sarcoma	R—X	Dead 1 yr.	
74 J.R.	18	F	0	2 yr.	0	Scalp	Spindle cell sarcoma	S—R	Dead 1 yr.	Node metastases
75 G.H.	23	M	0	6 mo.	2 excisions	Scalp	Spindle cell sarcoma	X	Dead 5 mo.	
76 F.L.	61	M	0	?	0	All over body	Melanosarcoma	X	No recurrence 2 yr.	Lung metastases
77 S.F.	40	M	0	3 mo.	0	All over body	Melanosarcoma	X	No recurrence 1 mo.	Lung metastases
78 G.S.	52	F	0	6 yr.	6 yr. resection. 1 yr. resection	Ischiorectal fossa	Fibrosarcoma	S—X	Well 7 yr.	

8. Attempts at complete surgical removal proved futile in all cases, probably because of poor encapsulation and interwoven connections with the neighboring structures.

9. Recurrences are characteristic of this form of sarcoma.

10. In the absence of gross or microscopic evidence of nerve structures, or nerve-producing structures, we are not willing to assign fibrosarcomas and spindle cell sarcomas to the neurogenic group.

11. In this series, 48 per cent were of the spindle cell and fibrosarcoma types.

12. Melanosarcomas with smaller, more diffusely pigmented cells proved more highly malignant than tumors composed of large cells with retained pigment.

13. Based on our findings, round cell sarcoma of soft tissue does exist as a histological entity.

14. No general plan of therapy can be advised for this group of cases, since, in every instance, the tumor and the patient must be individualized and treated accordingly.

15. Melanosarcoma proved to be the type of tumor resulting fatally earlier than the others.

16. Although eventually succumbing, patients with fibrosarcoma lived longest.

17. Myxosarcoma, similarly, was a radiosensitive, favorable type tumor.

18. Amputation was performed in too few cases to warrant arriving at a conclusion as to its value.

19. Spindle cell sarcoma, mixed cell sarcoma, and melanosarcoma are very prone to produce metastases; melanosarcoma disseminates generally throughout the body and especially to the liver and neighboring lymph nodes; spindle cell sarcoma tends to metastasize to the lungs, and to several odd sites; mixed cell sarcoma usually metastasizes to the lungs.

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DISCUSSION

Dr. Fred W. Stewart, New York City. My discussion of this paper will necessarily be from the pathological rather than the therapeutic viewpoint. First, I must take an exception to the statement that the term "neurogenic sarcoma" has of late come to be applied to those tumors which formerly were called fibrosarcoma and spindle cell sarcoma. I believe that the term neurogenic sarcoma should be employed only under certain conditions. These conditions may perhaps be listed as follows: (1) the patient has evidences of von Recklinghausen's disease either fully developed or in the forme fruste, with café au lait pigmentation and rare scattered cutaneous neurofibromas, either one or the other or both; (2) the tumor is anatomically dissected from a nerve or occurs within the nerve trunk; (3) the tumor shows the rather characteristic whorl structure seen in neurosarcoma and is unlikely from its gross anatomy a tumor of some well-defined structure such as muscle, periosteum, bone, fat, tendon or bursa. When a tumor shows the structure usually seen in neurogenic tumors

but where one cannot grossly demonstrate the connection, one can only diagnose provisionally and by inference. In this respect I might quote from one of Virchow's statements of 1863: "In all probability, therefore, the number of tumors which must be reckoned in the neuroma territory extends farther in many directions than one at present realizes."

Pathologists object to the diagnosis of neurosarcoma oftentimes because they say there are no nerve structures or nerve producing structures. That is readily understandable. If one studies the bulbous swellings of the nerves in von Recklinghausen's disease where there can be no question of the origin of the process, it is found that rather early in the edematous swelling the axones degenerate and in a series of nodules all stages of degeneration may be found with appropriate staining. Now the regeneration of neuraxones is not a peripheral process. Neither the sheath of Schwann nor the perineural nor endoneural connective tissue cells ever under any circumstances produce nerve fibers. Hence when either or both of these cell lines gives rise to a tumor one certainly should not expect it to produce nerves. The term "neurogenic sarcoma" then applies to a tumor originating from a nerve but not one producing nerves.

I believe it unfortunate that so many pathologists look in the microscope and so few see patients and do gross dissections. Certainly under many conditions the microscope does not tell us the origin of a tumor. Let me illustrate by three cases. In the first there is a bulky complex tumor of the thigh. In various parts of the tumor there is "round cell sarcoma," "fibrosarcoma," "xanthosarcoma." The microscope does not tell us they are neurogenic. Yet in serial operations on this same case we can trace this tumor back to its primary form and it is seen to have arisen in the classical edematous nerve trunk tumors of von Recklinghausen's disease. In another case the tumor showed rather characteristic whorls. That alone might lead us to suspect the origin but only an incidental dissection of the underlying nerves showed the proliferative activities of nerves of the neighborhood—in the nature of a neuroma. This patient had café au lait pigmentation. In the third case, the tumor was to all intents and purposes what you would call a fibrosarcoma yet it grew *within* the median nerve distending it, rupturing it, but with its upward extension still within the trunk, in vessels or lymphatics.

This does not mean that all spindle cell sarcomas of soft parts are neurogenic, but it does mean that you need more than the microscope to tell you that any particular spindle cell sarcoma is *not* neurogenic. Within the past week I have had a neurosarcoma of the upper tibia invading from a typical cirsoid neuroma of the nerves about the knee. This in itself is of interest but of more interest were two small nodules less than 2 mm. in diameter in the surrounding muscle. On dissection I could not find a nerve attached to these although from the accompanying evidence they are of the same type and they show a malignant structure. If one cannot find the nerve when the tumors are small one cannot expect to find it necessarily if they are large since the nerve itself does not necessarily enlarge with the tumor. At the time of my 1931 paper with Copeland we had more tumors of the neurogenic variety than of others. During the last two years we have had relatively few and are getting more tumors anatomically and histologically of muscle origin. In time our ratio of types may change in some other direction. We will not I hope go back to a pure histological terminology of round, spindle, giant cell, or fibrosarcoma.

I would be flattered if I thought I had been asked to discuss this paper from the clinical viewpoint. I am much interested in the treatment of these diseases but each year I think I know less and less. One thing may be said and that is that in my experience the group as a whole offers little in the way of enhancing one's self confidence in his therapeutic abilities either as a surgeon or a radiologist. I feel sorry for the people who have to treat them with the only methods now at our disposal, and among them Dr. Kaplan has perhaps my deepest sympathy for after everyone else gets through probably making things worse he has to take care of them.

DR. RUBENFELD (closing). We would rather not be too strenuous in refuting the use of the word "neurogenic" in connection with sarcomas, especially in view of the fact that Dr. Stewart has worked them up so carefully in his 1931 publication. However, from the clinical standpoint, we frequently encountered tumors from the thigh, some interpreted histologically as spindle cell sarcoma and others as fibro-

sarcoma, which behaved in an entirely different manner. As a rule, the fibrosarcomas proved to be much slower growing, although much more resistant to treatment, and rarely manifesting metastasis; entirely different from the spindle cell sarcoma which, as a rule, proved to be rapid in growth, much more difficult to enucleate under surgical dissection and often producing metastasis. From that alone, we believed we were handling two types of tumors and it was difficult to concede that they both arose from neurogenic or nerve structures.

There is one clinical point that we must stress as far as substantiating a neurogenic origin is concerned. After we made this study, we noted that recurrences did not take place in an excised area. On the other hand, there was usually a point of tenderness above or below the scar, apparently in the line of the nerve where recurrences appeared, which led us to assume that evidently that nerve was growing or being stimulated and irritated. Often directly in the tender site over the nerve a recurrent nodule appeared. Such a sequence would lend additional support to the clinical neurogenic sarcoma.

As far as therapy is concerned, we certainly take the same disheartening viewpoint that Dr. Stewart mentioned. We have tried in the last several years to give very intensive doses with the 5 gram pack. As yet the results are too recent to elaborate.

Recently, Dr. Handley, in a bulletin from the American Society for the Control of Cancer, suggested very radical excision of these tumor masses. If in the thigh, he advocates a radical removal down to the periosteum and excision of the nodes whether clinically involved or not. We have not tried that in the past, rather we have utilized massive doses of therapy. However, we believe the one point we could stress in this study is that a simple removal of a small, innocent-looking tumor which proves histologically to be sarcoma and then administering several doses of roentgen rays or radium is definitely inadequate treatment. Radical surgery, amputation if you will, followed by intensive doses of roentgen therapy may offer the better solution to this stubborn problem.

THE DANGERS OF REDUCING FRACTURES UNDER THE ROENTGENOSCOPE AND METHODS OF PROTECTION AGAINST THEM*

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THE use of the roentgenoscope as an aid in the reduction of fractures and in the removal of foreign bodies from an extremity is a common surgical procedure. Pendergrass, for example, has said:

> The advantages of a roentgenoscopic table for reduction of fractures are many, some of them are as follows: 1. The number of open reductions is lessened. 2. The number of times ether or local anesthesia is administered is decreased. 3. The economic advantage is of importance to the patient, surgeon, roentgenologist and hospital, because the fracture usually requires only one reduction, thereby taking less time and the convalescence is shorter and the hospital, therefore, has a quicker turnover of patients. 4. The number of roentgen-ray examinations is materially lessened. 5. Emergency cases of fractures may be more easily handled.

One of the earliest uses of roentgen rays of course was to visualize the bones in the human body. Pioneers in roentgenology soon recognized the dangers of excessive exposure to radiation. Imboden, for example, said that on account of some unfortunate experiences in the early days of roentgenology, Americans were very loath to practice roentgenoscopy. As a result the roentgenoscope did not come into general use in this country until about 1912.

As the knowledge of the possibility of injury from roentgenoscopy has become more common, many roentgenologists have come to agree with Carman that roentgen dermatitis is no longer a problem for the expert roentgenoscopist, it being uncommon to encounter a roentgenologist with a roentgen burn which he has received in the last five to ten years. It is not at all uncommon, however, to meet physicians, usually general practitioners or industrial surgeons, who have sustained roentgen dermatitis on the dorsum of their fingers as a result of prolonged or repeated roentgenoscopy in the course of reducing fractures or searching for foreign bodies. Chronic roentgen dermatitis of the hands from the use of the roentgenoscope is the most common skin injury seen among physicians.

It is usually accepted that burns due to irradiation are most often received by physicians who are careless or who are not thoroughly familiar with the clinical principles and use of roentgen rays. In spite of the widespread employment of the roentgenoscope by radiologists as well as nonradiologists, there are no articles in the literature of the last ten years directly referring either to the risks of reducing fractures under the roentgenoscope or to a method by which limits of safety might be determined.

The problems of protecting the physician's hands during removal of foreign bodies and during the reduction of fractures are not similar. In general, many principles apply in both cases, but the position of the operator's hands in the re-

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duction of a fracture differs from their usual position during removal of a foreign body. This is highly important in the consideration of exposure, for as will be shown later, great protection is afforded by the intervening extremity under examination. In fracture work it is seldom that the thickness of the extremity protects the operator's hands, since he usually keeps his fingers below the extremity and thus closer to the source of radiation. In the removal of foreign bodies the tissue being examined is almost always between the source of radiation and the operator's fingers. Because of these differences, this paper will be confined to a consideration of the problems of protection in the reduction of fractures only.

Recent radiological literature contains several articles dealing with the mathematics and physics of protecting the operator or patient against irradiation. But because of the highly technical nature of these articles, it would be extremely difficult for one not versed in the subject to understand them.

In a review of twenty well known textbooks on orthopedics and surgery we could find nothing directly referring to the dangers of roentgenoscopy or a method for determining the limits of safety. Nor do the companies which manufacture roentgenoscopes and advertise them to physicians warn of the possible danger from overexposure. The material pertinent to this paper has been obtained from outstanding articles on the subject of protection against overexposure and is presented here in an effort to show the nature of information available to, and understood by, the non-radiologist.

The handbook on "X-Ray Protection,"

TABLE I

FACTORS OF PROTECTION AGAINST OVEREXPOSURE TO ROENTGEN RAYS AS GIVEN BY VARIOUS AUTHORS

	Eliason	Hawley	U.S. Army X-Ray Manual	International X-Ray and Radium Protection Commission	U.S. Bureau of Standards	Advertising of Manufacturers
Gloves	*		**	***	***	
Hands under screen only when necessary	+			+		
Small area		+	+	+	+	
Adaptation of eyes to dark		No	+			
Short time of exposure		+		+	+	
"On-off" tube activation		+				
Timer clock		+				
Minimal intensities				+	+	
Lead glass in screen				+	+	+
Protective aprons and sheets					+	+
Aluminum filter					+	
Asbestos filter					+	
Number of reductions permissible			Two a week			
Cumulative effect			+			
Warning of late injuries			+			
Bone detail necessary?		No				
Alternate with assistant	+	+				
Hands out of field at all times		+				

* Two pair; kind not stated
** Surgeons' rubber
*** Lead-rubber

published in 1931 by the United States Department of Commerce, Bureau of Standards, contains some information pertinent to our problem. For complete details the reader is referred to this pamphlet. Some of the recommendations made in this pamphlet are listed in Table 1.

The United States Army X-Ray Manual, 1932, mentions the cumulative action of repeated exposure to radiation and warns of late appearance of injuries. The value of dark adaptation of the eyes as well as the use of rubber operating gloves is mentioned. Two roentgenoscopies per week is given as the limit of exposure for the operator.

One of the orthopedic surgeons who has contributed greatly to the literature on the roentgenoscopic reduction of fractures is Hawley. In a paper written by him in 1932 he said:

> At present the plan of action in a fresh fracture has been somewhat as follows: A man with a fracture of the tibia, for instance, is placed on the table. Traction is applied to the leg by means of the traction stirrup. Biplane radiographs are taken for permanent records and diagnosis. The fracture is reduced under the fluoroscope in daylight with the x-ray tube below. A nurse holds a box fluoroscope. The surgeon has his hands free and keeps his hands away from the fracture area. Reduction is made by regulating traction and by rotating and angulating fragments from the foot. This the surgeon does himself or in cooperation with an assistant. . . . Early trials indicate that a small fluoroscopic screen is large enough because the fracture area is small. A small screen leaves the sides of the limb exposed. It has been found that this fluoroscopic work can be done in daylight with a box fluoroscope. It is not necessary to wear a hood or accustom the eyes to the darkness except when fluoroscoping the region of the hip. Bone outlines show so sharply that the immediate accommodation of the eye is enough. Gross appearance is all that is necessary. The same fine bone and structural detail required in bone diagnosis is not necessary in reducing fractures. . . . With practice one can get accustomed to short repeated illumination of the screen. It is possible to install an automatic "on and off" switch in addition to the continuous foot switch. The operator then has his choice of either. To prevent overexposure in each case, an alarm clock with cut-off can be controlled by clock mechanism. . . . With care and thought it is possible to make reduction of fracture under the x-ray reasonably safe because of: The elimination of exposed high tension wires, the low milliamperage needed, the use of a small diaphragm and small screen, the confining of the scattering rays by the use of an adjustable cone from the tube box to the patient's leg, the exclusion of the surgeon's hands from the fracture area and from the direct rays, and the use of mechanical and instrumental manipulation by the surgeon in place of the surgeon's hands to effect reduction.

Certain statements made by Hawley may provoke controversial remarks from those doing fracture reductions. Carman has mentioned, for example, that many important details may be missed in roentgenoscopy if sufficient time is not allowed for adaptation of the eyes to the dark. Hawley was of the opinion that such adaptation was not necessary and that reduction of fractures can be done roentgenoscopically in daylight if the examiner uses a box roentgenoscope. The use of a small fluoroscopic screen may also be questioned by some, and the use of an alarm clock to cut off exposure after a certain time has elapsed is a very dubious means of preventing overexposure, for many factors besides time enter into a consideration of erythema, whether of the patient's skin or that of the operator's hands. Hawley stated that low milliamperage is used in his technique. Would it not be possible, by adaptation of the eyes to darkness, to use a still lower intensity?

Pendergrass wrote:

> In using the roentgenoscope, the tendency is to overexpose the patient and the surgeon. It is a rule in our laboratory that the intermittent control shall always be used in examining any patient with the roentgenoscope. That is, the roentgenoscope is used for a few seconds and then turned off for a second, thereby lessening the tendency to overexposure.

This suggestion has long been a subject of

controversy among radiologists. Some question this technique and prefer continuous activation of the roentgen tube.

In 1926, Eliason stated that roentgenoscopic reduction is used in as many cases as it is humanly possible, regardless of the time of day. Too much exposure is prevented by wearing two pairs of gloves and having the fingers under the screen only when necessary. He also recommended that an assistant should alternate in receiving exposure and that a roentgenologist should supervise the work.

Two recent textbooks on roentgenologic technique, one by Sante (second edition, 1935) and the other by Rhinehart (second edition, 1936) discuss in a general way the dangers of overexposure to roentgen rays. Sante stated that a valve-rectified apparatus is more dangerous to use than a mechanically rectified one because of the softer rays set up by the former. He also quoted the U. S. Army X-Ray Manual in stating a safe limit of exposure at any one time for a patient. The value of the aluminum filter, lower kilovoltages, small fields and increased distances are said to increase the amount of permissible exposure. The text makes no specific mention of the dangers of roentgenoscopy nor of the limits of safety in this procedure other than the citation from the U. S. Army X-Ray Manual.

Rhinehart states that all diagnostic apparatus should include a filter (thickness and kind not specified) and that no part of the operator's body should ever be placed in the direct beam of rays from a roentgen tube. No specific warning is made of possible dangers in the roentgenoscopic study of fractures or foreign bodies.

The suggestions which have been recommended for protection of the operator from excessive exposure to roentgen rays are listed in Table 1 in an effort to show the frequency with which these statements have been made. It will be seen from Table 1 that gloves of some sort are commonly recommended. However, only two of the five mention lead-rubber gloves. It is well known that rubber gloves having a lead equivalent of 0.5 mm. are a distinct help if worn for the protection of the hands. However, it is difficult to reduce a fracture with such heavy and cumbersome gloves and many physicians accordingly have not employed these gloves for the protection they afford. It will also be noted that a short exposure time and small diaphragm opening are recommended frequently, but only the U. S. Army X-Ray Manual mentions the value of "dark adaptation" of the eyes, only one of the references emphasizes the value of aluminum filters, and only one mentions the cumulative effect of radiation on the operator's hands and warns of the possible late appearance of injuries. Minimal intensities and proper lead glass in the fluorescent screen are mentioned but twice.

Offhand it seems that a table constructed along the general lines of the Hawley-Scanlan fracture table, in the design of which mechanical manipulation of the fracture is substituted for manual manipulation, might well reduce the risks of roentgenoscopic reduction of fractures. It is unfortunate that apparatus of this type is not available to all physicians who do roentgenoscopic reduction of fractures, but is confined almost entirely to hospitals and clinics doing a fairly large amount of orthopedic work. However, it may be difficult to use mechanical traction in the treatment of fractures of the bones of the hands or feet and of the very common fractures close to the ankle and wrist.

Advertising material regarding roentgenoscopes, as put out by three leading manufacturers of such apparatus, emphasizes complete shock-proofing. Occasionally some device for protecting the operator from radiation is mentioned in the advertisements. One of the concerns manufacturing roentgen apparatus in their description of a new model shock-proof roentgenoscope states:

> The operator's face is protected by a heavy sheet of clear lead glass, mounted in front of the fluoroscopic screen. Large lead shielded handles secure the operator's hands from exposure and

provide a safe means for controlling the movement of the fluoroscopic screen.

Another company, in describing their diagnostic tables, brings forth two valuable protective arrangements, namely, the limitation of the fluorescent image within the confines of the fluoroscopic screen at any table-screen distance and the complete enclosure of the under side of the table. It is of interest to note that this protective method was recommended by the U. S. Bureau of Standards in 1931.

A third roentgen-ray manufacturing company, in describing their roentgen tables, state that a cone is provided to reduce scattered radiation and that lead glass of proper thickness is provided in the fluorescent screen.

We realize that it is not the duty or function of manufacturers of roentgen equipment to teach the matter of protection to physicians who use their apparatus. However, nothing is mentioned in their literature on the possible dangers of roentgenoscopy, although much emphasis is laid on the fact that the roentgenoscopes are safe, shock-proof, and offer the utmost in protection. These statements may mislead the physician without roentgenologic training into thinking that he is safe from overexposure or he may think that since overexposure is not mentioned it does not make any difference how long he runs the machine.

It is remarkable and astonishing that the roentgenologic and orthopedic literature of the last ten years mentions so little with regard to specific warnings of the possible dangers of roentgenoscopic reduction of fractures or how these dangers may be avoided. The amount of radiation that one may safely receive over a long period of time has been estimated by several investigators, but there is some doubt as to the actual value of these doses. Leddy, Cilley and Kirklin have shown that the roentgenoscopists at The Mayo Clinic have greatly exceeded these "tolerance doses" over a period of years and to date have not shown any signs of injury from roentgen rays, which would tend to throw some doubt on the validity of these ideas as to tolerance doses. It is not the purpose here, however, to discuss the so-called tolerance doses which have been suggested. They are probably safe, and for the time being should be accepted.

We thought it more advisable to determine doses in terms of a fairly uniform constant, namely, the "erythema" dose rather than the "tolerance" dose. With physical factors such as are used in roentgenoscopic work, the majority of the roentgenologists and dermatologists agree that about 375 to 400 roentgens at one exposure is the erythema dose.

It is obvious from what has been stated so far that nothing much is known, or at least written, about the risk of reducing fractures under the roentgenoscope. Therefore it was decided to investigate the problem with the following points in mind: (1) to determine the approximate time required to produce erythema of the operator's hands under the conditions met with when reducing a fracture with the aid of the roentgenoscope, (2) to ascertain the advisability of protecting the operator's hands, (3) to find, from the standpoint of protection, an optimal combination of kilovoltage, milliamperage, and filtration to be used in reducing the commonest fractures, such as those of the arm, leg, hand, foot, wrist and ankle, (4) to investigate the protective value of various roentgenoscopic table tops, and (5) to make certain suggestions, if indicated by our results, which might help to prevent these serious injuries of the skin which are so common.

It is obviously impossible to employ the exact technical factors employed in the reduction of each individual fracture. Therefore, we have investigated a standardized technique which we feel best suits average conditions. This consisted of 80 kv., 6 ma. of tube current, 16 inch (40.6 cm.) tube-target-table distance, and a table top of $\frac{1}{4}$ inch (0.64 cm.) pine veneer.

A standardized mechanically rectified roentgen therapy apparatus with an air-

cooled Coolidge tube (Fig. 1) was used because of the danger of ruining the ordinary roentgenoscopic tube by the prolonged use this study would necessitate. As has been stated, 80 kv. and 6 ma. were used. Eighty kilovolts is a fair average of the voltage usually employed in clinical procedures. Six milliamperes is in excess of the average tube current used, but correction can easily be made for this. The distance between the tube-target and the top of the table was 16 inches (40.6 cm.). The table top was made of ¼ inch three-ply pine veneer.

THE DOSE TO THE OPERATOR'S FINGERS

The first problem was to determine the amount of roentgen radiation received by the back of the operator's fingers. It is obvious that when one grasps a limb in preparation for reduction of a fracture, one's fingers are invariably below the extremity and one's thumb is on the side or top. In this test the intensity of the roentgen rays was measured with an ionization chamber, which was placed on the table where the fingers would be placed during reduction of the fracture. Measurements of the intensity of the roentgen rays were made without a filter, with a filter of 1 mm. of aluminum, and with one of 2 mm. of aluminum, a Victoreen r meter being used. The center of the ionization chamber was placed in the center of the roentgen-ray beam and 1 inch (2.54 cm.) above the top of the table to allow for thickness of the operator's fingers. The tube, as has been said, was run at 80 kv. and 6 ma.

Since we are using 375 roentgens as the amount of radiation necessary to produce erythema, it must be remembered that the roentgens can be measured only in air according to definition, while in this study the intensities were measured with an intervening table top, or table top and human extremity as "secondary radiators." The Victoreen r meter, which had recently been standardized at the factory, was found to be very accurate with the radiation we used. The same beam of radiation when measured first by a Victoreen and then by a Mekapion roentgenometer gave almost complete agreement, but because of the ease of handling it was preferred to use the Victoreen instrument in this study. In all the measurements the same roentgenometer was used which, with the "secondary radiators" held constant during the experiments, minimized the error in measurements of dosage.

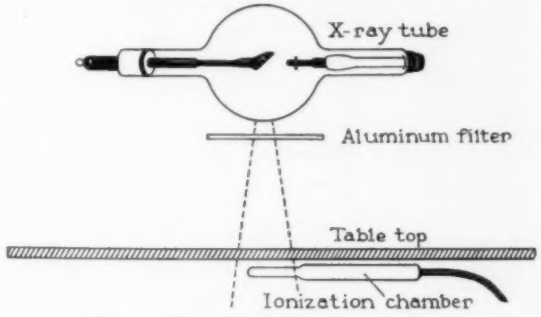

Fig. 1. Details of apparatus used.

Under the foregoing conditions, it was determined by ionization chamber readings that the erythema time was seven minutes and forty-five seconds without a filter, fifteen minutes and thirty-seven seconds with a filter of 1 mm. of aluminum, and twenty-four minutes with a filter of 2 mm. of aluminum. These figures were obtained by determining the time for the ionization chamber to discharge 25 r with each system of filtration, and then multiplying this time by fifteen so that the time for 375 r could be ascertained. The initial readings were checked repeatedly and on different days. At no time was an error of more than 5 per cent noted.

It must be kept in mind that 80 kv. and 6 ma. were used in these determinations. It also should be remembered that, in general, with other factors constant, the intensity of radiation is directly proportional to the milliamperage, that it is inversely proportional to the square of the distance between the tube-target and fingers, and that it is directly proportional to the square of the kilovoltage and decreases with filtration.

Others often use a technique in actual

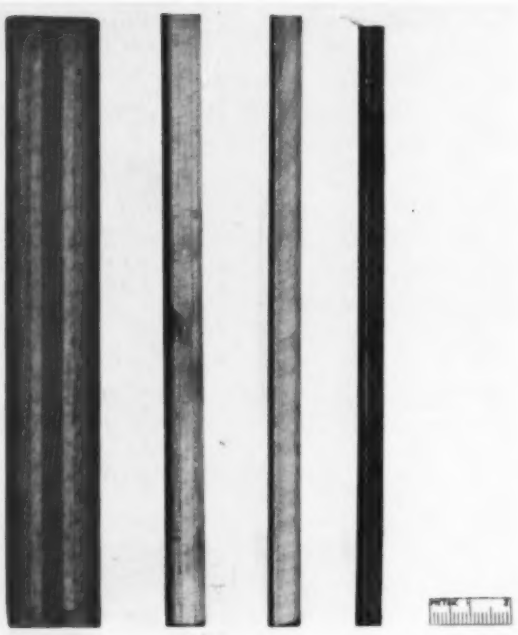

FIG. 2. Various standard roentgenoscopic table tops. From left to right: Company A's, Company B's, Company C's ($\frac{3}{4}$ inch) and Company C's ($\frac{1}{4}$ inch).

TABLE II

DOSE OUTSIDE OF CENTRAL RAY TO DORSUM OF OPERATOR'S FINGERS

Filter	Position of ionization chamber outside of central ray, inches	Erythema time	
		Minutes	Seconds
None	2	7	30
None	4	8	15
None	6	8	52
1 mm. Al	2	14	30
1 mm. Al	4	16	
1 mm. Al	6	17	37
2 mm. Al	2	22	52
2 mm. Al	4	24	6
2 mm. Al	6	28	15

practice that varies from these standard conditions; nevertheless it is easy to estimate the erythema dose for each individual roentgenoscopic technique by using these factors as a basis of calculation.

The Dose Received by the Index Fingers. It usually happens that the index fingers are more severely injured than the other fingers from reducing fractures under the roentgenoscope. This is probably because the index fingers lie nearest to (or in) the central ray. The data in Table II show that as one moves the hand out of the central ray, the doses received by the fingers become successively less; they also show the advisability of keeping the fingers out of the central ray.

THE EFFECT OF THE TABLE TOP ON THE "DOSE"

Next, the influence of the thickness of the table top was investigated. Samples of roentgenoscopic table tops as at present marketed were obtained from three manufacturers of roentgen-ray equipment (Fig. 2).

The table top of Company A "is 7/8 inch thick and made of five layer ply-wood, and covered on both top and bottom with 1/8 inch bakelite." It was claimed in the advertising pamphlets that this table top allowed unobstructed transmission of the roentgen rays both for roentgenography and roentgenoscopy.

The table top of Company B was made up as follows: "The upper surface is of bakelite 3/64 inch thick, ply-wood of three layers totaling 16/64 inch, is then coated with an aluminum sheet 0.0005 inch thick which tends to disperse static electricity and act as a soft ray filter."

The two table tops of Company C according to specifications had the following construction: "The $\frac{1}{4}$ inch board is made of 3/16 inch three layer ply-wood with a 1/16 inch bakelite cover, and the thicker top, 3/8 inch is made of five layer ply-wood 5/16 inch thick, with a 1/16 inch bakelite top panel." The thinner board is used in the vertical roentgenoscopic tables, and the thicker one in their other tables.

The table top serves two purposes: (1) to support the patient, and (2) to filter the beam of radiation. All the table tops investigated were built of ply-wood and bakelite. In addition to this the Company B top contained a thin sheet of aluminum on the tube side.

The filtration value of each table top was

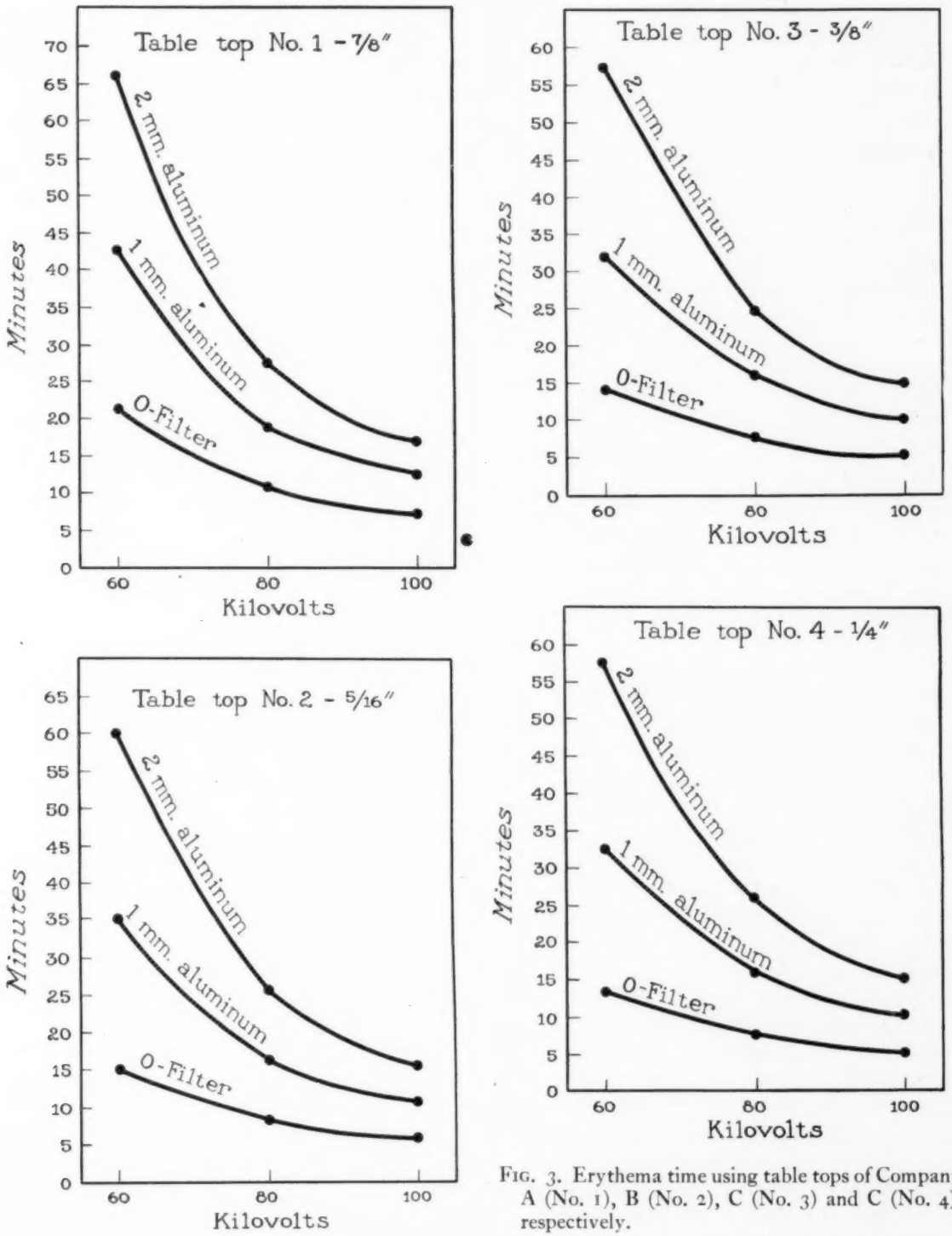

FIG. 3. Erythema time using table tops of Company A (No. 1), B (No. 2), C (No. 3) and C (No. 4), respectively.

determined by the use of the Victoreen r meter. A uniform tube-target-table-top distance of 16 inches (40.6 cm.) was maintained and the ionization chamber was placed 2 to 3 mm. above the table top and in the central ray. Six milliamperes was used with varying kilovoltages, and aluminum filters of varying thickness were used.

TABLE III

ERYTHEMA TIME FOR THE DORSUM OF THE OPERATOR'S FINGERS USING VARIOUS TABLE TOPS, 6 MA. OF TUBE CURRENT, AND 16 INCH TUBE-TARGET-TABLE-TOP DISTANCE (ERYTHEMA DOSE = 375 r)

Company	Kilovolts	Erythema time, minutes		
		No filter	Aluminum filter	
			1 mm.	2 mm.
A	60	21¼	42½	66
	80	11	19	27½
	100	7½	12½	17
B	60	15	35	60
	80	8½	16½	25½
	100	6	10½	15½
C (¼ in.)	60	13	32½	57½
	80	7¾	16	26
	100	5	10	15
C (¾ in.)	60	14	32½	57½
	80	7¾	16	25
	100	5½	10¼	15

The erythema time for each set-up was determined as before. Table III shows the filtration value of the several table tops and how the erythema time varies with the different table tops, and Figure 3 gives these results in graphic form. It seems to be largely a matter of thickness rather than material which determines the protective value of each respective table top. At first glance, the few minutes' difference in erythema time between a thin and thick table top may seem to be of no consequence, but when it is remembered that doses of roentgen rays are cumulative, then these few minutes' difference in erythema time over a period of years of roentgenoscopic work will become of great significance, particularly since the radiation through the thinner table top is softer and accordingly is more completely absorbed in the skin.

From the foregoing one may conclude that table tops have a definite although slight protective value. This value may be entirely discounted if an equivalent amount of aluminum filter is added to the apparatus. However, in certain instances when no table top is used, as with the Company A table, there is added danger because the operator's hands can get closer to the tube without intervening protection of any sort.

The value of aluminum filtration as a protective device is well shown in Table III. In certain cases, such as with the Company A table, there is nothing intervening between the roentgen tube and the extremity being examined. Naturally, with other factors constant, the erythema time will be less in these cases and the danger of overexposure of the operator increased. Using ordinary pine ply-wood the actual protective value of two thicknesses of this material was determined in order to show the erythema time both with and without table-top protection. These data will be found in Table IV.

THE EFFECT OF SECONDARY RADIATION

Secondary radiation from the extremity being examined under the roentgenoscope does not play a great part in the total "dose." Theoretically, the secondary component of the dose should not be large because of the low voltage which induces it, coupled with the fact that the irradiated volume is small. Actually it is not large, and the weak secondary rays from the limb are absorbed in the palmar aspect of the

TABLE IV

VALUE OF TABLE TOP IN PROTECTION OF THE OPERATOR'S FINGERS

Table top	Filter	Erythema time, minutes	
		With table	Without table
¼ inch 3-ply pine veneer	None	7½	6¼
	1 mm. Al	15½	14½
	2 mm. Al	23¾	22½
¾ inch 5-ply pine veneer	None	10¼	6¼
	1 mm. Al	17½	14½
	2 mm. Al	27½	22½

hand and fingers. Further evidence that these rays are weak is the fact that roentgen dermatitis is very rare on the palmar aspect of the operator's hand. However, the precaution of keeping the exposed field as small as possible is well worth observing on general principles.

THE DOSE RECEIVED BY THE THUMB OF THE EXAMINER

Before determining the dose received by the thumb when it is on the top of the limb, there was some question as to whether the absorption characteristics of a wax or other phantom, with the radiation in question, were not different from the absorption characteristics of human tissue. We therefore carried out all measurements on the forearm of the cadaver of an adult (Table v), which forearm was of average adult dimensions. It was placed on the table top and the ionization chamber was set from 1 to 2 mm. above the skin.

The figures in Table v show the great amount of protection afforded to the operator's thumb on the top of the forearm under examination by the tissues of the forearm, in comparison to the dose received by the fingers on the under side of the extremity. The erythema time was so long at the two sites chosen that it was not thought necessary to take readings with the greater thickness of the tissue of the arm or thigh.

Table V

RELATION OF THICKNESS OF TISSUE TO ERYTHEMA TIME

Thickness of tissue, inches	Filter	Erythema time, minutes
1½ at wrist	None	56+
1½ at wrist	1 mm. Al	75
1½ at wrist	2 mm. Al	93+
2½ at upper part of forearm	None	100
2½ at upper part of forearm	1 mm. Al	128
2½ at upper part of forearm	2 mm. Al	156

Table VI

ERYTHEMA TIME IN MINUTES WITH VARYING VOLTAGE AND FILTRATION

Kilovolts	No filter	Aluminum filter		
		0.5 mm.	1 mm.	2 mm.
50	16¼	30	46¾	83½
60	11¼	18¾	27	47½
70	8½	13¾	20	31½
80	7½	10¾	15	23½
90	5½	8¾	12	17½
100	5	7½	10	15

THE DETERMINATION OF AN "IDEAL" ROENTGENOSCOPIC TECHNIQUE

The next step was to determine a combination of kilovoltage and filtration which would afford the operator more protection from excessive radiation than the techniques commonly used and yet give good visibility of the fracture. By using kilovoltages varying from 50 to 100, and aluminum filtration varying from 0.5 to 2 mm., a ¼ inch ply-wood table top, a 16 inch tube-target-table-top distance and 6 ma. of tube current, the erythema time for each set-up was determined (Table vi).

From the information given in Table vi the great amount of protection afforded by an aluminum filter is well shown. This is especially true at the lower kilovoltages. Roentgenoscopes should have easily accessible filters. Many radiologists say that varying conditions met with during roentgenoscopy demand various types of penetrating roentgen rays. This can be governed by the electrical factors to a great extent, but an intelligent use of filtration will afford more protection in each instance. The figures in Table iii show how, by varying the kilovoltage and filters to meet the existing conditions, more protection to the operator can be obtained. Many roentgenoscopes have the filter, if they have any at all, in an almost inaccessible place, and hence once a filter is in position it is seldom changed. If arrangements could be made whereby the filtration might be easily changed, it would be of benefit to

vary the filter to obtain the longest erythema time in combination with the optimal visibility of the tissue being examined.

Objections may be made that this increase in erythema time will lead to carelessness, or that with changeable filters all filtration will be forgotten. However, if the operator remembers that he is dealing with something that can predispose to cancer of his fingers, he will not likely forget the matter of filtration.

Five members of the Section on Roentgenology of The Mayo Clinic were asked into a room equipped with a standard-type horizontal roentgenoscope that they might test some theoretical results. They were given twenty minutes to adapt their eyes to the darkness. One of us examined roentgenoscopically the fractured forearm of a cadaver, using technical factors unknown to those making the test. The men were then asked to write their opinion of the visibility and detail of the bony structure as brought out by various combinations of kilovoltage and filter. Throughout the test the tube-target-table-top distance was 16 inches and the tube current was kept constant at 3 ma. The arm was placed on the top of the "horizontal table" which was of $\frac{1}{4}$ inch pine veneer. Each person kept his opinion secret during the test. After the experiment was over the results were tabulated and these are shown in Table VII with comments in the roentgenologists' own words.

It may be seen from Table VII that 80 kv. with no aluminum filter was regarded as giving too bright a field. Eighty kilovolts with 2 mm. of aluminum as filter was considered to give, in general, the same roentgenoscopic results as the former. Seventy kilovolts with 1 mm. of aluminum filter was also considered as giving too bright a field. Fifty kilovolts with varying filter provoked numerous comments, but all were in agreement that any of these 50 kv. set-ups would be satisfactory for visualization of a fracture of the arm or leg.

It would seem from the foregoing opinions that an optimal setting for roentgenoscopic reduction of a fracture of the forearm or leg would be about 55 kv. with 2 mm. of aluminum as filter, and 3 ma. of

TABLE VII

DETERMINATION OF "IDEAL" TECHNIQUE

Roentgenologist	A	B	C	D	E
1. 80 kv., no filter	Too bright	Good	No detail too bright	Fair	Too bright
2. 50 kv., no filter	Not quite so bright	Not quite so good as Number 1	Better	Good	Good
3. 50 kv., 2 mm. Al	Bone detail not so bright	Fair	Fair	Good	Good
4. 70 kv., 1 mm. Al	Too bright	Fair	Too bright	Good, bright	Too bright
5. 50 kv., 0.5 mm. Al	Bone dark	Same as Number 2	Good	Good	Good
6. 80 kv., 2 mm. Al	Almost like Number 1	Not quite so good as Number 1	Too bright	Too bright	Too bright

Note: 3 ma. of tube current and a 16 inch tube-target-table-top distance were kept constant.

tube current, if both the visibility of the fracture and protection of the operator are considered the deciding factors.

As has been shown in Table VI, the erythema time to the dorsum of the operator's fingers when they are placed on the table top is forty-seven and a half minutes when the following factors are used: 60 kv., 6 ma. of tube current, 2 mm. of aluminum as filter, $\frac{1}{4}$ inch pine veneer table top and a tube-target-table-top distance of 16 inches. With 3 ma. of tube current, the erythema time would be ninety-five minutes.

Several other radiologists were asked what factors they used in roentgenoscopy (of all types). The average kilovoltage employed by them was about 80, and the majority of them used from 0.25 to 0.5 mm. of aluminum as a filter. They varied the milliamperage and not the kilovoltage with the part of the body being examined. From Table VI it may be seen that with 80 kv., 0.5 mm. of aluminum filter, and 6 ma. of tube current, the erythema time is about ten and three-fourths minutes; with 3 ma. the time would be doubled. Our "ideal" technique of 55 kv., 2 mm. of aluminum as filter, 3 ma. of tube current, 16 inches tube-target-table-top distance, and a table top of $\frac{1}{4}$ inch pine veneer, allows more than five times the amount of protection to the operator's hands, even when they are in the direct field of radiation, and yet permits perfect visualization of the fracture.

Before summarizing this paper it should be emphasized that no attempt has been made to present something new to the expert radiologist, but rather to investigate the problem from the standpoint of protecting physicians who are doing roentgenoscopic reduction of fractures. Many practicing physicians, interns, and residents who use a roentgenoscope are not aware of the actual dangers of overexposure and have little knowledge of the actual physical and electrical factors they employ. It should be repeated that the factors used in our experiments will most likely vary from those used in other roentgenoscopic techniques, but they represent what is believed to be a fair average. These values may be used in estimating the doses received by others. The figures in this paper cannot be used as absolute unless the working conditions of others are identical to those in this work.

SUMMARY

With 80 kv. and 6 ma. of tube current, with 16 inches between the tube-target and the table, and with a $\frac{1}{4}$ inch pine veneer table top (conditions closely approaching those in actual practice), it was found that the erythema time to the dorsum of the operator's fingers on the table top in the center of the field of radiation during reduction of a fracture with the aid of a roentgenoscope is seven minutes and forty-five seconds without any filter, fifteen minutes and thirty-seven seconds with 1 mm. of aluminum as a filter, and twenty-four minutes with 2 mm. of aluminum as filter.

The erythema time increases as the fingers are withdrawn from the center of the field of radiation.

There is no marked difference in the protective value of any of the samples of actual table tops examined. If one considers that the doses of radiation received in roentgenoscopy are cumulative, then the thicker table tops are a distinct help in protecting the operator's hands. Aside from thickness, the construction and materials used in the table top do not seem to be of any great importance, providing they contain no metal.

A table top between the roentgen tube and the fracture area is of definite protective value.

In comparison with the radiation received on the dorsum of the fingers, the thumb is well protected by the thickness of the limb which is fractured.

A suggestion has been made of those factors which will give the best visibility and at the same time the best protection.

There are in the literature no specific warnings or recommendations in regard to roentgenoscopic reduction of fractures.

CONCLUSIONS AND RECOMMENDATIONS

It cannot be emphasized enough that roentgenoscopic reduction of fractures of the extremities is a very dangerous procedure. The busy or careless roentgenoscopist may, in the course of a single reduction of a fracture, receive a severe injury to the dorsum of his fingers. He may also receive radiation below an actual erythema in quantity, which later, by cumulation, may give rise to injury of the skin. The patient at the same time may receive an erythema dose (or even an excessive dose) which may be masked by the pain of the fracture and which may not be detected because of inflammation following fracture, especially since the limb may be enclosed in a cast.

This study emphasizes the importance of speed of examination, short activation of the tube, keeping the fingers outside the central beam of rays, and of using the protection afforded by the thickness of the extremity being examined, factors which make for safety in all roentgenoscopic procedures.

The standardized technique used in this study may be used as a basis for calculating doses actually received when the roentgenoscopic technique is different.

It is recommended especially that the hands of the operator be kept out of the field when the roentgen tube is activated, particularly if lead-rubber gloves are not worn, and reduction of fractures under the roentgenoscope is strongly condemned unless the operator carefully measures the doses he receives, or unless he is under the direct supervision of an experienced radiologist, or unless he uses *all* recommended protective devices.

A technique consisting of 55 kv., 3 ma. of tube current, 2 mm. of aluminum as filter, 16 inches tube-target-table-top distance, and a table of $\frac{1}{4}$ inch pine veneer may not be ideal but, with modifications according to clinical expediency, may be worthy of trial.

It is suggested that roentgenoscopic apparatus have readily changeable filters, and mechanical manipulation of fractures, as suggested by Hawley, in place of manual reduction is well worth consideration.

Since the effects of roentgen irradiation are cumulative and evidence of injury may develop late, it is recommended that each operator keep a permanent record of the radiation he has received on his hands from all roentgenologic examinations. As an additional safety factor, the operator should calculate his maximal possible exposure and consider that he has received this dose even though additional protective factors may have been employed.

It is suggested that the various committees on roentgen-ray protection make their recommendations from a clinical as well as physical point of view, which they have not done to date.

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Vol. 37, No. 1

A DOSAGE SYSTEM FOR RADIUM THERAPY

PRELIMINARY REPORT

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THE following investigations were prompted by a desire to be able to express the amount of radiation received by a tumor or a tissue as a fraction or percentage of the amount of radiation given off from a radioactive source.

The term intensity is used to express density or concentration of the rays at a point in the field of radiation; the term dose refers to milligram-hours at the source or milligram-hours per square centimeter on a skin surface. Milligram-hours gives a more convenient figure for the present purpose than millicuries-destroyed.

In Figure 1, suppose the dose at the point source P is 10,000 mg-hr. A certain fraction of the radiation, perhaps 0.01 per cent, reaches the tumor T. In other words, the tumor receives the full effect of 0.0001 × 10,000, or 1 mg-hr. As long as the tumor receives the full effect of 1 mg-hr., it makes no difference to the tissue from what distance the radiation comes nor indeed whether the radiation originates inside or outside the tumor. Obviously, the enormous reduction in intensity is the effect of distance and filtration. A simple formula is sought which will express the fraction of

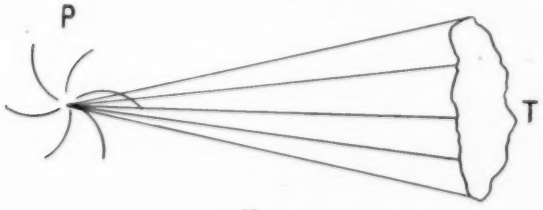

FIG. 1

radiation transmitted through any thickness of filter to any distance.

The effect of distance alone can be calculated with greater theoretical than practical precision and the results of the calculations require no experimental verification.

A point source gives off radiation equally in all directions. Imagine a sphere of radius r drawn about a point source of radiation R, as in Figure 2. The surface area of the sphere is $4\pi r^2$ and all the radiation coming from the source R passes through the surface. If the dose at R is 10,000 mg-hr., the

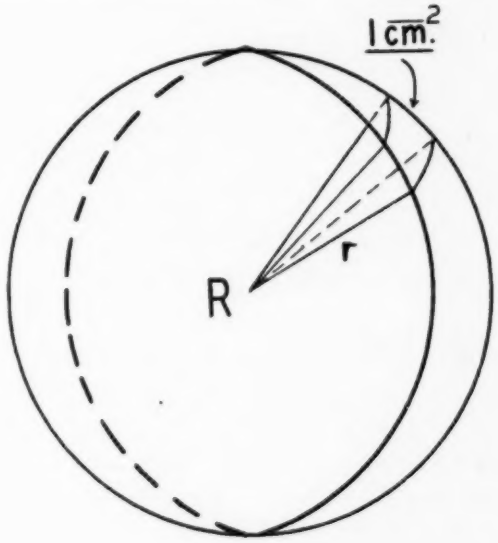

FIG. 2

dose on the surface of the sphere is also 10,000 mg-hr., but the surface contains $4\pi r^2$ sq. cm. Therefore, the dose on each square centimeter is $R/4\pi r^2$ or $795/r^2$ mg-hr. If r is 10 cm. the dose on the surface is 7.95 mg-hr. per sq. cm. In other words, in this particular case every square centimeter receives the full effect of 7.95 mg-hr. If the square centimeter is a square centimeter of tissue (a skin surface, for instance) it will make no difference to that square centimeter of skin from what distance or direction the rays come so long as it receives the full effect of 7.95 mg-hr. Thus, the dose of radiation reaching a surface from a point source can be expressed by a simple relation

$$D=\frac{R}{12.566r^2} \qquad (1)$$

where D is the dose received in milligram-hours per square centimeter, R the dose at the source in milligram-hours and r the distance in centimeters.

Still ignoring the filtration or assuming it to be constant, suppose the radiation required to produce an erythema has been found from observation to be 7.95 mg-hr. per sq. cm., then the dose required at the source at any distance is found from the formula $R=D12.57r^2$. If D is 7.95, then $R=(7.95)(12.57)r^2=100r^2$. The value for r is of course selected according to the depth intensity or depth dose desired.

The effect of filtration alone cannot be calculated with theoretical accuracy and any formula to express the percentage or fraction of radiation transmitted through a filter must be developed from experimental data.

The intensity of a monochromatic beam of radiation passing through a filter is expressed by the familiar equation

$$I=I_o e^{-ux}$$

where I is the transmitted intensity, I_o the original intensity, u the coefficient of absorption and x the thickness of the filter. Calling the initial intensity 100 per cent and putting

$$\frac{I}{100}=y$$

we have

$$y=e^{-ux}$$

where y is the fraction of the radiation transmitted. Since gamma rays vary in wave length this formula as it stands cannot be used. Experimental determinations of y for various thickness of filter indicate that the absorption formula for radium rays has the general form

$$y=ce^{-kx^n}$$

where c, k and n are constants depending on the character of the filter. The problem now is to determine the value of these constants.

Table 1 shows the intensity of radiation transmitted through various thicknesses of lead, the intensity expressed both as percentage (I) and as a fraction (y) of the original intensity. The source used consisted of bare glass tubes of radon and may be considered a point source. The values are not corrected for scattering.

TABLE I

x mm. Pb	I per cent	y fraction	$-\log y$
0.43	3.67	0.0367	1.435
0.86	2.75	0.0275	1.56
1.074	2.645	0.02645	1.578
2.157	2.222	0.0222	1.653
3.25	1.99	0.0199	1.701
6.74	1.44	0.0144	1.842
10.25		0.01165	1.934
13.78		0.00935	2.03
17.28		0.00779	2.108
20.8		0.00654	2.184
24.33		0.00553	2.257
28.64		0.00456	2.341

Let us observe the effects of plotting values of x and y in various ways using y as ordinate and x as abscissa. Plots on plain graph paper or on semi-logarithmic paper give smooth curves which drop rapidly from $y=1$ and then gradually approach zero. However, if the values are plotted on logarithmic paper the curve is S-shaped as in Figure 3 (AB). The first part of the curve, i.e., in the left upper quadrant, is evidently due to gamma rays plus beta rays, while the remainder of the curve is due to gamma rays alone. The continuation backward of that part of the curve due to gamma rays alone would obviously go along the dotted line C, eventually reaching some definite value of y where $x=o$ (when $x=o, y=c$). We now wish to find the value of c. If x is plotted with $-\log y$ on logarithmic paper (D) the curve is flattened out so that one can almost guess the value of $-\log y$ as x approaches zero. It can be calculated with some degree of accuracy, however, and is found to be 1.427 which makes $c=0.0374$, or 3.74 per cent.

This value 3.74 evidently represents the

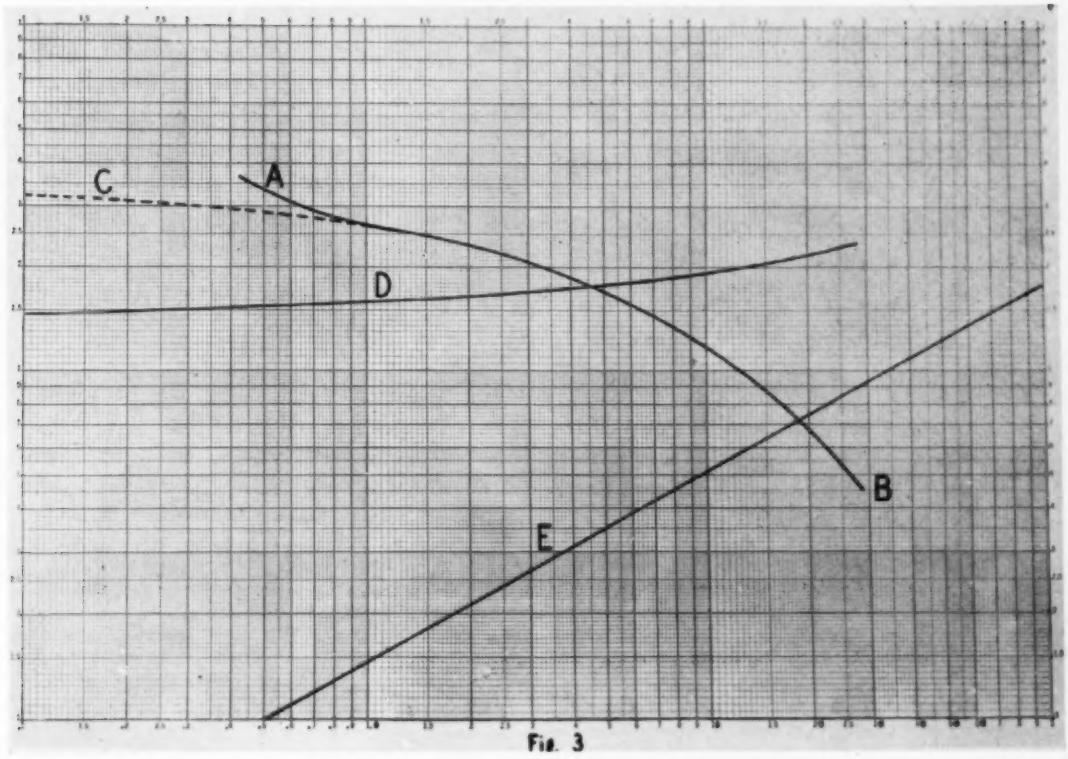

Fig. 3

percentage of gamma rays in the radiation from a bare glass tube of radon. Calling this intensity 100 per cent for gamma rays, we then form a table in which $y/0.0374$ or y' is the fraction of gamma rays transmitted (Table II).

Now if the values of x and $-\log y'$ are plotted on logarithmic paper a straight line is obtained (E, Fig. 3). Using all the points to find the formula for this line

$$\log(-\log y') = \log k + n \log x$$

$$-0.883 = 5 \log k + 6.131\, n$$
$$-3.285 = 5 \log k + 1.640\, n$$

$$2.402 = 4.491\, n$$
$$n = 0.53485$$

$$-3.285 = 5 \log k + 0.887$$
$$\log k = -0.8344 = 9.1656 - 10$$
$$k = 0.14652$$

$$-\log_{10} y' = 0.14652 x^{0.53485}$$
$$-\log_e y' = 0.337 x^{0.535}$$
$$y' = e^{-0.337 x^{0.535}} \qquad (2)$$

This represents the fraction of gamma rays transmitted through any thickness of lead (in millimeters) and to find the transmitted intensity in percentage the result is simply multiplied by 100. If it is desired

Table II

x	y'	$-\log y'$
0.86	0.735	0.134
2.157	0.594	0.227
6.74	0.385	0.415
10.25	0.3115	0.507
20.80	0.175	0.757
28.64	0.122	0.914

to find the intensity of total radiation transmitted $y/0.0374$ is substituted for y' whence

$$y = 0.0374 e^{-0.337 x^{0.535}} \qquad (3)$$

By combining (2) or (3) with the distance formula we can find the amount or intensity of radiation reaching a surface at any distance from a point source of radon

and through any thickness of lead. For gamma radiation alone

$$y' = \frac{R}{12.57r^2} \cdot e^{-0.337x^{0.535}} = \frac{0.07958Re^{-0.337x^{0.535}}}{r^2}$$

or for total radiation

$$y = \frac{0.00298Re^{-0.337x^{0.535}}}{r^2}$$

the result being expressed as milligram-hours per square centimeter. Since y' may be considered the erythema dose and values of x and r are selected according to the quality of radiation and depth intensity desired the quantity to be found is usually the amount of radiation required at the source.

$$R = 12.57y'r^2e^{0.337x^{0.535}}.$$

The purpose of this preliminary report is to present a method rather than a strictly correct result. For this reason many factors have been left out of consideration. The value 3.74, for instance, depends on the wall thickness of the glass tubing employed. The average value is probably somewhere between 3.5 and 5.5. The measurements given above are not corrected for scattering. The lead employed is probably not chemically pure. More accurate measurements are now being made with other metals (Au, Pt, Ta, Cu, Al, etc.). Perhaps some definite relation can be found between the density or atomic number of the heavy metals and their rate of absorption so that a separate formula will not be necessary for each metal.

SUMMARY

A method is presented whereby the dose of radon received by a surface from a point source can be calculated as a definite fraction or percentage of the total amount of radiation given off at the source. The dose is expressed as milligram-hours per square centimeter. A type formula is presented which gives the dose of gamma rays falling on a square centimeter of surface at any distance from a point source and through any thickness of lead,

$$y' = \frac{0.07958Re^{-0.337x^{0.535}}}{r^2}$$

where R is the dose of radiation at the source in milligram-hours, x the thickness of lead in millimeters, r the distance in centimeters, and y' the milligram-hours per square centimeter.

To determine the average intensity over a surface from a point source or at a point from a non-point source, the formula can be integrated. This will be discussed more fully in a later publication.

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LOST AND FOUND RADIUM*

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EVERYONE who uses radium element in any form is faced with the possibility of loss, which loss is of much more concern than the loss of a similarly valuable particle of other precious material, as the radium has the potentiality of terrible damage to any uninformed person who may come in contact with it.

As radium, when used for medical purposes, is invariably associated with contaminated dressings, it is only reasonable that this material is rapidly removed from its place of use, to be thrown out with the trash and often incinerated.

The purpose of this paper, aside from being a collection of serious or ludicrous instances (depending on the point of view), is to show that radium has been lost in many instances and that the chance of recovery is extremely high. The fact that so many diversified kinds of apparatus were used in the many hunts tends to show that if some ingenuity is used and a careful analysis is made of the situation, practically anyone who is familiar with the properties of radium has an excellent chance of locating it.

My first experience with misplaced radium came in 1933 when I had used 75 mg. in tubes and needles in the treatment of a superficial malignancy of the hand. Through some mistake, my technician believed that I had replaced the radium in the safe, while I believed that she had done so. On the following morning the loss was discovered and I at once remembered that the patient had brought his two small children into my office, both of whom had shown much curiosity in the radium. My unhappy conclusion was that the children had liked the appearance of the containers and carried them along for toys. As the patient lived out of the city and out of access by telephone, the matter seemed grave. Fortunately the trash for the previous day had not left the premises and a small electroscope with spark coil charger[1,2] was rushed out into the back yard. Within fifteen minutes the radium was back in the safe, but it had been a long fifteen minutes and we had learned a lesson.

FIG. 1. Simple electroscope with spark coil charger.

Some time after that I arranged the electroscope in a container with the charger, which made a fairly compact and reliable little instrument.[3]

In the winter of 1935, I received a frantic telephone call from Monroe, North Carolina, to come and search for two 12.5 mg. needles that had been used about three weeks before, and from all information been replaced in the safe. Two reliable nurses had seen it put in the safe but it was no longer there. I delayed my departure long enough to build four more electroscopes and put them in a carrying case large enough to hold other materials,

* Read at the Thirty-sixth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 24–27, 1935.

FIG. 2. Radium detector incorporating electroscope and charger.

though I did not have time to mount the charging devices with them. I arrived in Monroe about dark and after removing the remaining radium checked the safe, all rooms, halls and drain traps, as far out as the place where the trash was deposited, and found no trace of the radium. The municipal dump seemed the next place, so dawn found me there. The weather was cold, with puddles of water frozen and many parts of the dump afire. With the cooperation of the city manager, we determined that the last three weeks' refuse was probably confined to an area about 40× 200 feet. We arranged to griddle this area with the electroscopes, placing one every 8 feet on a cross line, allowing them to remain five minutes, then moving the line forward about 6 feet and repeating. The alignment was greatly facilitated by lines of cord with chrome yellow flags tied at the required points. The work was slow and difficult, as we were working down the side of an embankment and through many areas of fire or water, scrap iron and brambles. Despite the fact that many of the instruments were often covered with ashes and dew, they all functioned well. Many times they were put in tin cans to protect them. At about noon two of the instruments showed some drop of the leaf. These were removed to test for natural leak and other instruments substituted for them. When the second set showed a fall, we began to get interested. All of the instruments were bunched around but no definite localization could be made, which proved that the radium was buried some distance below the surface. With the aid of colored boys who brought shovels and cardboard boxes, the material consisting mostly of cinders and leaves was taken out and removed a distance of about 50 feet, my assistant checking the boxes on their arrival, while I checked the bottom of the hole. After several cubic feet had been removed, we got two boxes in succession which gave strong indications and the hole went "dead." By dividing the contents of the boxes, it was a simple matter to isolate the needles.

Within one week after this experience, I was called to a city in a neighboring state on a similar mission. In this case the amount of radium lost was not definitely known, and the time at which it was last used was not determined, as no hospital chart could be found and the estimated dates varied from seven to three months previously. During that time the portion of the hospital in which the radium had been used had been rebuilt, all of the city dumps had been flooded by the adjacent river and much of the refuse scattered. After one day of work the search was aban-

FIG. 3. "Field kit" which was hurriedly constructed to carry eight electroscopes and necessary equipment.

doned. This is an excellent example of the use of radium by those who have no respect for it.

Knowing that the loss and recovery of radium, although invariably news, is not uncommon, I have collected the following incidences from personal communications with my colleagues:

Dr. Curtis F. Burnam—Amount of radium lost or the date of the loss is unknown. One tube misplaced on hospital dressing. Tube recovered from city dump with electroscope.

Dr. Albert Soiland—75 mg. of radium lost, date unknown. Patient slipped away from nurse and deposited radium, concealed in waste paper, behind soil pipes in toilet. Radium located by means of electroscope.

— 2 or 3 mg. radium tubes lost, date unknown. Radium discarded on adhesive dressing. Recovered quite by accident in a waste receptacle in the basement of the building.

Mr. J. L. Weatherwax—50 mg. radium applicator lost in 1930. Recovered by electroscope immediately in ashpile which had gone through incinerator.

—10 mg. radium needle recovered in a like manner in 1931.

—200 mg. applicator lost in 1932. Recovered from dump by means of electroscope.

—40 mg. applicator lost in 1935. Radium had gone into the hopper, was recovered in last trap of drain in basement, just before going into sewer (Geiger counter).

—10 mg. needle lost in 1935. Radium located on floor of hospital by Geiger counter.

—8 or 10 mg. needles lost, date unknown. Electroscope located radiation but could not get definite source, as radium had been crushed and broken.

—10 mg. radium needle lost, date unknown. Radium had been lost six months. Never located.

—50 mg. applicator, lost 1932. Located within hospital by electroscope.

Dr. G. E. Pfahler—100 mg. radium lost about 1931. Located on city dump, after much searching.

Dr. Otto Glasser—600 mc. (radium emanation bulb) lost in 1922. Traced and found that it had been discarded with bandages and probably gone through furnace. No portable electroscope available, so had two colored boys bring ashes on shovels close to the standard electroscope. When the fiftieth shovel was brought forth, the unharmed tube was pulled out, much to the boys' surprise.

Fig. 4. A typical view of municipal dump, Monroe, North Carolina, showing character of the material in which the work was done.

—Radium salt lost in 1923. Went to furnace and in examining the ashes found that the tubes must have been destroyed, as the radioactive substance was scattered. Located by means of an electroscope.

—Radium tubes lost in 1924. Located unharmed in ashes from furnace (electroscope).

—10 needles containing radium salt lost in 1927. Patient walked out of office with needles in bandage. Radium located the next morning scattered on floor and staircase of hospital (electroscope).

—5 mg. radium needle lost in 1934. Located with Victoreen r-meter.

—Radium emanation tube lost in 1935. Located with the Victoreen proteximeter.

About 1932 a loss of about 35 mg. of radium was sustained in Chattanooga, Tennessee, and never recovered, with a similar occurrence with 5 mg. about 1929. In both instances, the radium was probably thrown on the city dump. Methods of search, if any, are now not known.

Mr. F. L. Curtiss of the Bureau of Standards has designed a very ingenious instrument on the vacuum tube principle for the purpose of locating radium.[4] While the stated sensitivity is rather low, this difficulty is well compensated for by the complete portability of the machine and the fact that an immediate reading is given by a microammeter, instead of the customary method of waiting a period of minutes for an effect to be observed. The Bureau of

Standards reports that this instrument was successful in locating 25 mg. of radium which had been on a city dump for three days. The recovery was accomplished in less than twenty minutes. This occurred in September, 1934.

A somewhat similar instrument has been designed by the British National Physical Laboratory.[5] It is said to work on the principle of amplification, by means of a neon lamp.

A rather amusing example of the ridiculous statements of the popular press occurred when the *Radio Guide* stated that a young operator of Station WSM found $3,000 worth of radium, with an instrument made of some sulphur and a bread pan. This interested me and I communicated with the operator, only to find out that he, Mr. A. E. Omberg, Physicist for Vanderbilt University, had very cleverly used a Victoreen r-meter connected to a large ionization chamber. He subsequently published an account of this.[6]

An insurance company that handles this risk has given me the following:

Losses as a result of fire are probably the least hazardous from the viewpoint of total losses, because, as we understand it, radium is more or less indestructible. Under extreme circumstances it may become slightly contaminated and/or fused with débris, but can be reclaimed without any serious consequences. Sometime ago we were informed of an incident where, through carelessness, a substantial quantity of radium reached a hospital incinerator but was subsequently all recovered from the ashes. In another similar occurrence, but before the search had started, the cinders were sold to a contractor and finally traced to having been used as a base under a new cement walk. The radium was actually located there with the assistance of a highly scientific detector but the cost of retrieving the same, including the expense already incurred to trace the radium to this point, would have exceeded the value of the radium.

The theft hazard is undoubtedly always prevalent, although we have no knowledge of any radium robberies. There have been losses in the mail, we having sustained one amounting to approximately $1000.00, fortunately at a time when radium was only valued at $20.00 per milligram.

There are two instances in which radium was probably flushed through the sewage system, one in Chattanooga and one in Savannah; in both instances 50 mg. of radium was lost. As both of these cities are located on a river, the radium probably reached the ocean where most treasures have been lost throughout history and where they will probably remain until better methods of salvage are devised.

The most spectacular radium recovery of all time occurred in Sioux Falls, South Dakota, on August 28, 1935. Having checked the newspaper account by a personal communication from Dr. J. W. Buchta and found it correct, I quote as follows:

An amazing detective story relating how two university sleuths went searching for a tiny radium capsule and recovered it from the stomach of a pig was revealed here today.

The impromptu detectives were Dr. J. W. Buchta, of the physics department and Dr. H. H. Barber, of the chemistry department, both of the University of Minnesota. The scene of their spectacular discovery was Sioux Falls, S. D.

The radium, valued at $3000.00, was contained in a tiny capsule three quarters of an inch long and barely one-sixteenth of an inch in diameter. It was used by physicians at the Moe Hospital in Sioux Falls for treatment of cancer. A nurse unknowingly discarded a piece of adhesive tape to which the capsule clung.

Hospital authorities, when they discovered the capsule gone, easily learned how it had become lost. From there they could go no farther, so they called Dr. Barber and Dr. Buchta.

The "sleuths" went immediately to Sioux Falls. Their equipment consisted of an ash can in which was built an upright rod. A gold leaf was suspended from the bottom of the rod. Electricity in the rod made the leaf stand out at right angles. The presence of radium would overcome the electric power and flatten the leaf against the rod.

Like true detectives, the two professors started at the beginning and learned that rubbish from Moe hospital was taken to a pig farm operated by F. L. Tibbles.

At the farm they set up their instrument. There was no quiver from the leaf, so they moved around over various rubbish piles. Sudenly the leaf shook and flattened against the rod. Radium, the detectives knew, was nearby.

A moment later, however, without the instrument having been moved the leaf showed no reaction. This puzzled the sleuths until one of them observed that a herd of swine had been walking past. Then Tribbles, who had been watching the entire proceeding with his tongue in his cheek, took an interest. He divided his 500 pigs into five groups and the test was repeated near each group. One of them caused the leaf to flutter. Again they divided the pigs into groups and by constant elimination one solitary porker, when held near the instrument, caused the reaction. The professors concluded they had found their pig.

A butcher was called. A moment later the radium was recovered.

A count of the above instances shows that radium has been lost on twenty-three occasions (total amount lost 1,488 mg.) with complete recovery of the radium uninjured, while in eight other losses (total 232 mg.) recovery was not effected.

Appreciation is expressed to those who kindly supplied information on their personal experiences.

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DISCUSSION

Dr. J. H. Vastine, Philadelphia. Dr. Taft's paper has been most interesting. His ingenious devices are familiar to all of you, and, as you know, have been most helpful in various fields of radiology. One cannot appreciate the importance of the convenient portable electroscope, as designed by him, for the detection of lost radium until one experiences the loss of some of this valuable element.

There were 100 mg. of radium lost from Dr. Pfahler's office several years ago, and this was eventually found by means of an electroscope. It was found 2 feet beneath the surface of the city dump, which covers a 10 acre field.

While discussing the means of finding radium, it might also be well to mention briefly measures to prevent its loss, and I will therefore briefly discuss such measures taken in Dr. Pfahler's office. The radium is checked every night by two people who sign for it, thus making them responsible. All dressings for each day are kept in a bag which is dated. The bags, which are put in a sanitary can, are kept for six weeks which gives one the opportunity of checking the dressings in case any radium is missing. The only radium loss occurred as a result of a group of circumstances including an unusually busy day, a rushed nurse, miscounting the radium, combined with a new maid giving the dressings to the trash collector rather than storing it in the basement.

As an additional precaution against permanent loss, an electroscope built for us by Dr. Taft is kept in the radium preparation room so that in case radium is misplaced, an immediate search can be instituted.

Mr. J. L. Weatherwax, Philadelphia. Dr. Taft's collection of data of lost and found radium was interesting and enjoyable. I brought along a Geiger counter with the idea of demonstrating to you how it detects radium. This was developed by Dr. Gordon L. Locher, associate of Dr. Swann, of the Bartol Foundation of the Franklin Institute. It is the same instrument that has been used to measure cosmic rays. Dr. Swann and Dr. Locher have both engaged in this work in balloon ascensions.

(Demonstrating) We have here the Geiger counter and also, back of it, an amplification system. This counter registers the clicks we get by the radium or cosmic rays. Cosmic rays will produce ten to twenty clicks per minute without any radium being present. I have here 2.5 mc. of radium emanation, or radon, which makes an impression on the Geiger counter at quite a distance away. I have an old emanation tube which we have discarded, and which also activates the counter. For all practical purposes, the life of this emanation tube is spent. I have here, too, an old dollar watch, not running, and I think you can get the detection of

this small amount of radioactivity from the face of the watch. This Geiger counter can be used for detecting weak radiation intensities and there is a possible field for such an instrument in detecting the stray radiation around the laboratory. We have our radium emanation plant on the first floor of our Laboratory Building, and some radiation can be detected on the third floor. This counter is surrounded by lead which makes it directional. I have used this instrument twice to locate radium. In most cases, in locating radium, I have used the electroscope because the counter was not available. Dr. Locher designed this in March, 1934. He wanted to detect a very small amount of radium that had accidentally been lost, and so built this instrument at that time. Some of you may have read a few months ago that an instrument like this had been developed in England, and claims were made that this was the first instrument of of its kind.

DR. OTTO GLASSER, Cleveland, Ohio. I would like to point out that the counter which we demonstrated downstairs in the scientific exhibit was built five years ago and since that time one of its uses has been the detection of radium. We presented it before the American Society for the Advancement of Science at its meeting in Cleveland in 1930.

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A COMPARISON OF PARAFFIN AND WATER PHANTOMS FOR ROENTGEN-RAY DEPTH DOSE MEASUREMENTS

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IN ORDER to study the distribution of radiation within the body, it is necessary to use some sort of organic material as a "phantom" within which the ionization chamber (or other measuring device) can be placed. The most generally accepted measurements of this sort have been made in water, and it has been shown that depth doses actually obtained in the human body agree with those made in a water phantom.[1]

From time to time the statement appears in the literature that paraffin is a satisfactory substitute for water. It is certainly easier to work with. But its density is about 0.9 and its absorption coefficient definitely less than that of water.[2] It has been noted that published depth doses obtained with paraffin phantoms are higher than those obtained with water phantoms.[3,4] It therefore seems important to make a comparison of the two under the same experimental conditions.

The roentgen rays used in this work were generated at 200 kv. (peak), with mechanical rectification, and filtered by 0.5 mm. copper and 2.5 mm. aluminum; the half-value layer was 0.8 mm. copper. The same spherical celluloid ionization chamber was used as in the work with the human cadaver;[1] it has a wall thickness of 1 mm., and a volume of 1.54 c.c. It is mounted on a slender brass stem, is water-tight, and needs no extra shield for use in the water phantom. Ionization measurements were made with the Failla vacuum tube apparatus.

The water phantom is a cylindrical aluminum tank, 37 cm. in diameter and 32 cm. deep. At no time is the ionization chamber less than 15 cm. from the sides and bottom of the phantom (either for water or paraffin). The paraffin phantom is made of a pile of circular blocks of high melting point paraffin (density 0.88), 30 cm. in diameter and 3.1 cm. thick. In two of these, depressions are cut so as to fit tightly about half of the ionization chamber and its stem, the chamber lying at the center of the circle. With one of these above and one below the chamber, it is completely enclosed, with practically no air space about either chamber or stem. These two blocks can be placed anywhere in the pile, or, for surface measurements, the upper one can be removed.

With a target-surface distance of 70 cm., depth dose measurements were made in the paraffin phantom for fields of 50, 100, and 400 sq. cm., the surface position being with the chamber half buried in paraffin. Water was then substituted, and measurements made for the same fields, the surface position being with the chamber half submerged. A typical set of results is given in Figure 1. The upper pairs of curves give the actual readings of the measuring instrument, the lower, these values reduced to percentage depth doses.

In the curves representing the actual readings, all values for paraffin are higher than the corresponding ones for water. The difference of about 4 per cent at the surface represents an actual difference in back-scattering. The difference at the depths would be expected to be due to the combined effect of differences in back-scattering and in absorption. With increasing depth, the difference between the two sets of results becomes more marked, so that at the depth of 5 cm. the paraffin gives depth doses about 9 per cent higher than the water, and at 10 cm., about 29 per cent higher. It is interesting to observe that

TABLE I

A COMPARISON OF RADIATION DELIVERED IN WATER AND PARAFFIN PHANTOMS

I	II	III	IV	V	VI	VII	VIII	IX	X	XI
Depth Cm.	Volts I_t	Inverse Square Factor	Distance Factor I_p	Abs. Factor δ_m	Primary Radiation $I_p \times \delta_m$	Scattering I_s	Percentage Scattering	Ratio Paraffin / Water	Percentage Depth Dose	Ratio Paraffin / Water
400 sq. cm.—Water										
0	3.12	100	2.23	1.00	2.23	0.89	29	1.06	100	1.00
2	2.90	93.0	2.10	0.685	1.44	1.46	50	1.02	93	1.02
5	2.30	87.0	1.94	0.389	0.76	1.54	67	1.01	75	1.10
7	1.87	82.6	1.84	0.267	0.49	1.38	74	1.01	60	1.13
10	1.27	76.5	1.71	0.151	0.26	1.01	80	1.02	41	1.29
12	0.92	72.8	1.62	0.103	0.17	0.75	82	1.02	30	1.43
400 sq. cm.—Paraffin										
0	3.25		2.23	1.00	2.23	1.02	31		100	
2	3.08		2.10	0.715	1.50	1.58	51		95	
5	2.63		1.94	0.432	0.84	1.79	68		81	
7	2.25		1.84	0.309	0.57	1.68	75		68	
10	1.72		1.71	0.186	0.32	1.40	82		53	
12	1.40		1.62	0.134	0.22	1.18	84		43	
100 sq. cm.—Water										
0	2.69		2.07	1.00	2.07	0.62	23.2	1.06	100	1.00
2	2.39		1.96	0.685	1.34	1.05	44	1.02	89	1.03
5	1.77		1.80	0.389	0.70	1.07	59	1.01	66	1.08
7	1.37		1.71	0.267	0.46	0.91	66	1.01	51	1.14
10	0.83		1.58	0.151	0.24	0.59	71	1.02	31	1.29
12	0.55		1.51	0.103	0.15	0.40	73	1.03	21	1.48
100 sq. cm.—Paraffin										
0	2.75		2.07	1.00	2.07	0.68	24.7		100	
2	2.51		1.96	0.715	1.40	1.11	45		92	
5	1.95		1.80	0.432	0.78	1.17	60		71	
7	1.58		1.71	0.309	0.53	1.05	67		58	
10	1.10		1.58	0.186	0.29	0.81	73		40	
12	0.85		1.51	0.134	0.20	0.65	76		31	
50 sq. cm.—Water										
0	2.33		1.90	1.00	1.90	0.43	18.4	1.15	100	1.00
2	2.03		1.80	0.685	1.23	0.80	39.5	1.04	87	1.04
5	1.49		1.65	0.389	0.64	0.85	57	1.02	64	1.09
7	1.12		1.57	0.267	0.42	0.70	63	1.02	48	1.16
10	0.65		1.46	0.151	0.22	0.43	66	1.04	28	1.30
50 sq. cm.—Paraffin										
0	2.41		1.90	1.00	1.90	0.51	21.2		100	
2	2.19		1.80	0.715	1.29	0.90	41		91	
5	1.68		1.65	0.432	0.71	0.97	58		70	
7	1.34		1.57	0.309	0.48	0.86	64		56	
10	0.88		1.46	0.186	0.27	0.61	69		37	

these differences are practically the same for the three fields studied.

It is evident from these data that measurements made in a paraffin phantom cannot be applied to depth doses within the human body unless a correction is applied. A correction factor can be empirically determined from the data, to be applied to measurements in a paraffin phantom, *for radiation of the quality here used, and at 70 cm. distance.* Without a detailed investigation of the origin of this correction factor, it is impossible to state whether it may be more widely used.

The method of analysis of data of this type developed by Thoraeus,[5] and more recently used by Parker,[6] may be applied to yield interesting information. This method separates the measured radiation into components due to the direct beam and to the scattered radiation, so that they may be compared. The complete analysis is presented in Table I. I_t (column II) denotes the total radiation intensity as measured by the ionization chamber. It is here tabulated in terms of volts read on the meter of the measuring instrument, with the phantom in place. (The values used are taken from smooth curves drawn through the experimental points.) Column III gives the factor for decrease in intensity due to increase in distance from the source with increasing depth, as calculated by the inverse square law. This is the same for every area, and for both phantoms. I_p (column IV) denotes the intensity of the primary beam as affected by the distance alone. The first value in this column, for each group, is the actual reading of the ionization instrument, with the phantom removed; the others are this first value multiplied by the proper distance factor from column III.

δ_m (column V) represents the part of the primary radiation which would pass

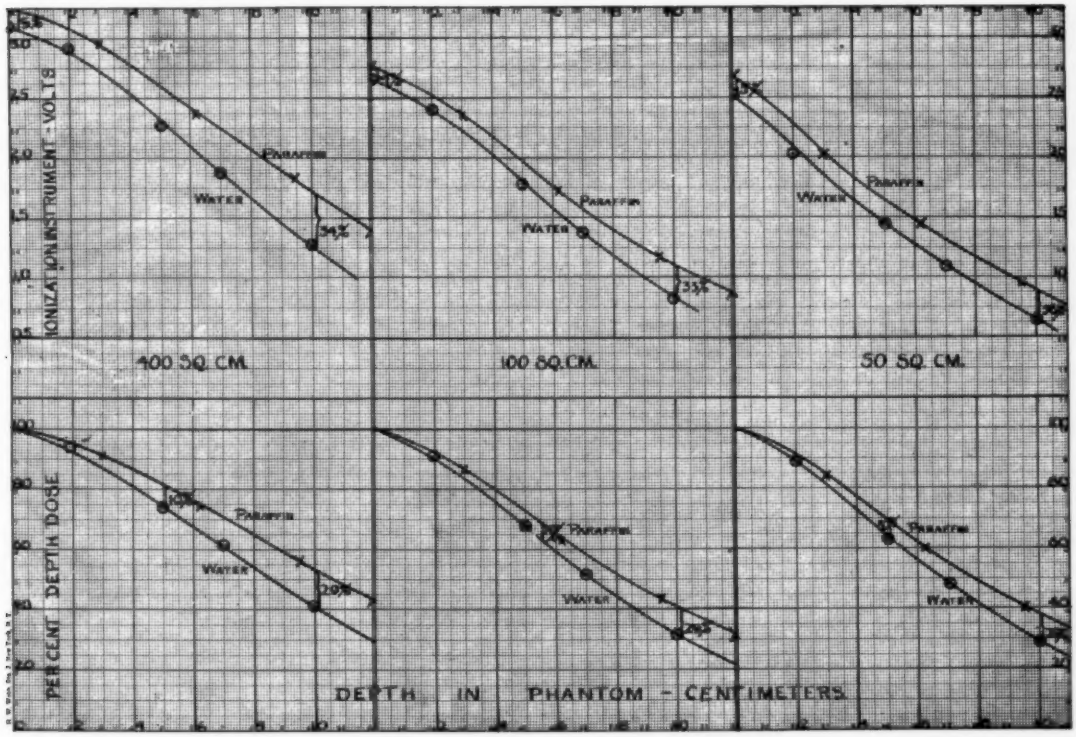

Fig. 1. Roentgen-ray depth dose measurements in paraffin and water phantoms. Upper series of curves give actual readings on ionization measuring instrument; lower series give percentage depth doses calculated from values of upper curves.

through the given thickness of water or paraffin, the diminution being due to absorption only (no scattering). This factor has not been experimentally determined for the present investigation. Thoraeus[3] gives values for δ_m for water, for the quality of radiation used here (0.8 mm. Cu half-value layer). These agree closely with those of Grebe and Nitzge[2] for water for radiation of wave length 0.16Å. Accordingly, the absorption coefficients for this wave length, for both water and paraffin, taken from this table, are used in the calculations. (μ_{water} = 0.189 per cm.; $\mu_{paraffin}$ = 0.168 per cm.) Of course, the radiation actually used is not monochromatic, but the ratio of the coefficients for water and paraffin remains fairly constant between 0.12Å and 0.20Å, so that the relative values based on these factors should give informative data.

In column VI, the product of I_p and δ_m gives that part of the primary radiation which arrives at the various depths, the two factors accounting for distance and absorption. This product, subtracted from I_t, gives that part of the total radiation which is due to scattering (I_s, column VII). The percentage of the total radiation at any depth, which is due to scattering, is found by dividing the values of I_s by those of I_t, the results being given in column VIII.*

It is interesting to note that, for the most part, the percentage of the total radiation due to scattering is almost the same for water and for paraffin. In column IX are given the ratios of the percentage of scattered radiation in paraffin to that in water, for all depths. Except for the surface values, the variation is not more than 4 per cent in any case, and the average is 2 per cent. Even the surface values for the large fields differ by only 6 per cent. In the surface readings for smaller fields, where the absolute value of the scattered radiation is small in comparison to the total radiation, a very small variation in the observed value produces a large percentage difference. An increase of 1 per cent in the surface reading for the paraffin, and a decrease of the same amount in the corresponding reading for water, would bring the figure of 1.15 down to 1.07. The measurements as made may contain errors of this magnitude. For no other pair of readings would a corresponding error cause a change of more than 2 per cent in the values of column IX. These values should, then, be accepted as indicating a slightly higher percentage of scattered radiation in the paraffin phantom, the effect being more marked near the surface.*

Column X gives the percentage depth doses, taken from the curves of Figure 1, and column XI the ratios of depth doses in paraffin to those in water. A comparison of these values with those in column IX shows that the much greater depth doses in paraffin cannot be attributed to the scattered radiation in the body of the phantom. It is evidently to be attributed to the difference in absorption of the primary radiation. It is for this reason that the ratio between paraffin and water values is the same for all fields; for the same reason, the difference between the two phantoms should be independent of the target-phantom distance.

Four factors enter into the calculation of the depth dose, as seen in the formula:

$$\text{Depth dose} = \frac{\text{Prim. rad. at depth} + \text{sec. rad. at depth}}{\text{Prim. rad. at surf.} + \text{sec. rad. at surf.}}$$

From the data of Table 1 it is evident that, for paraffin and water, for radiation within the quality range here studied, the corresponding terms differ by different de-

* It should be noted that the scattering is given as percentage of the *total* radiation. Values for back-scattering at the surface are usually given as percentage of the *primary* radiation, in which case they are, of course, higher.

* This surface effect may be due to the type of ionization chamber used, and this factor would also affect the relative depth doses. The actual depth doses, however, should be correctly measured with any small "air-wall" chamber of the type employed. Since the relative scattering values here given are based on the actual ionization readings in the depths, and not on percentage depth doses, any error inherent in surface readings does not invalidate the conclusion reached, that the percentage of the depth dose which is due to scattered radiation is practically the same in both water and paraffin phantoms.

The small but constant percentage of increase for the paraffin cannot be explained on the basis of experimental error. Neither can it be explained on the basis of a small error in the absorption coefficient chosen, for the coefficients used were for radiation of the same quality. A small change in one would necessitate a change in the same direction in the other, and the net result would be the same.

grees. The ratio between the numerator terms, for water and paraffin, varies with the depth, but the ratio of the first terms steadily decreases, while that of the second increases for the first few centimeters and then decreases. The first terms in the denominators are identical; the ratio between the second terms in the denominators is constant for a given beam. This relationship being so complicated, it is evident that no simple correction factor can be applied to readings in a paraffin phantom to give the values which would be obtained in water.

In view of these findings, it appears that a paraffin phantom should not be used for the determination of depth doses to be applied to therapy. The experimental results are not directly applicable, and cannot be readily corrected. Beeswax (density 0.97) or mixed waxes with unit density, while more expensive, are also more accurate. Parker[6] has found measurements made with his unit density wax to agree with those made in the water phantom.

The above statements must not be construed as condemning the use of paraffin phantoms for the comparison of various physical or biological set-ups, so long as the values obtained are not to be transferred directly to clinical use. Even in such work, however, the data would be more valuable if obtained with a scattering material of approximately unit density.

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VARIATIONS IN THE INTENSITY AND QUALITY OF ROENTGEN-RAY OUTPUT AS AFFECTED BY THE ANGLE TO THE CENTRAL RAY, THE ROENTGEN TUBE AND THE ROENTGEN MACHINE

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KAYE[3] in 1909 reported on the angular distribution of roentgen-ray intensity as determined from a gas roentgen tube operated at a comparatively low voltage. With the presentation of the hot cathode tube, Coolidge and Kearsley[2] also published curves for its angular intensity distribution in the plane containing the tube axis and perpendicular to the target face; these data were taken at 200 kv. Bouwers and Diepenhorst[1] have reported on the intensity of roentgen rays as a function of the angle of emergence from the surface of the anticathode, contributing both calculated and measured values. Their work at 70 kv. was more in the diagnostic range but was taken around the girdle of the tube and was from a 45° target. Because none of these data quite fit the modern diagnostic conditions met with in the line-focus, auto-protective tubes, the experiment herein presented was undertaken.

THE EXPERIMENTAL CONDITIONS

The following apparatus was used in this experiment: Victoreen thimble chamber roentgenometer; a line focus, 6 kw., Metalix* roentgen tube (focus 2.9 mm.); a line focus, 10 kw., Media roentgen tube (focus 3.9 mm.); and assorted commercial roentgen-ray filters of aluminum, whose actual thickness was measured by a micrometer. The high tension supply was from a four-valve-tube-rectified, 20 kw. generator, known as a Wappler Quadrex.

"Origin" is defined as the intersection of the tube axis with the focal plane of the anode. This is assumed to coincide with the center of the focal spot area. "Central ray" is defined as that ray, arising from the origin, which is perpendicular to the tube axis and to the focal plane of the anode. It is assumed that the central ray passes through the center of the portal of the auto-protective tubes. Angles are indicated as from the origin and to the central ray in the plane of the central ray and the tube axis. For convenience, a negative angle is designated as one taken in the direction of the cathode end of the tube, and a positive angle as one in the direction of the anode, from the central ray.

The roentgen tube was supported on a regular tube stand and adjusted each time so that its axis was accurately vertical as determined by a spirit cross-level. It was pointed in such a way that the nearest material object in the emergent beam, aside from the parts of the supporting cone itself, was 5 feet distant. This positioning was adopted because preliminary experiments had indicated sufficient back-scattering to introduce a source of error when the central ray was pointed downward toward a table upon which the roentgenometer was placed. The roentgenometer was positioned with its long axis perpendicular to the central ray and its center the specified distance from the axis of the tube.

To obtain readings at various angles to the central ray, the roentgen tube was shifted from the position where the center of the roentgenometer chamber intercepted the central ray, the vertical distance "b," such that the desired angle $\theta = \tan^{-1} b/a$,

* The trade name of the apparatus available for this experiment is given merely to define the conditions and without prejudice for or against either the product used or that of any other manufacturer.

or $b = a \tan \theta$, where "a" was the distance from the ionization chamber to the tube axis.

All measurements were made at a control panel setting which gave a reading of 60 kv. (prereading meter), and at 8 ma., with the time varied so that approximately the same displacement of the roentgenometer was obtained at each exposure condition. The filament was manually controlled during the exposure to maintain constant milliamperage; no way was available to insure non-variation of the potential so those exposures during which a definite change in the primary voltage was noted on the meter were discarded. Up to ten readings were taken at each setting, depending upon the length of exposure and the agreement between duplicate exposures, and the results were averaged. For a long exposure time, transient fluctuations would have small effect upon the results obtained and so only from one to three readings were made, but for short periods more were used.

Filters were added in the filter slots of the tube stand with their plane normal to the central ray. Thus the actual thickness of filter material at angles greater than zero degrees was more than the nominal thickness and was obtained by dividing the latter value by $\cos \theta$.

The values of intensity obtained by direct reading were reduced to intensity at a uniform distance from the origin by dividing them by the factor $\cos^2 \theta$. Values on a plane surface (such as a film) parallel to the tube axis were obtained by multiplying the values "as read" by $\cos \theta$. Values of r/ma-min. as given in this paper are those reduced to uniform distance unless otherwise stated.

I. THE OUTPUT AT VARIOUS ANGLES TO THE CENTRAL RAY

The Intensity at Various Angles to the Central Ray. The intensity of unfiltered radiation at 25 cm. from the origin from the Metalix tube is plotted on polar coordinates in Figure 1. Because the thimble chamber roentgenometer employed in these measurements is not accurate for use with the soft radiation obtained without filtration[4] and because the quality of radiation is different at different angles to the central ray, these results do not have absolute significance. They are only roughly quantitative and should be interpreted solely as indicating that the intensities of unfiltered radiation may be quite dissimilar at different angles to the central ray.

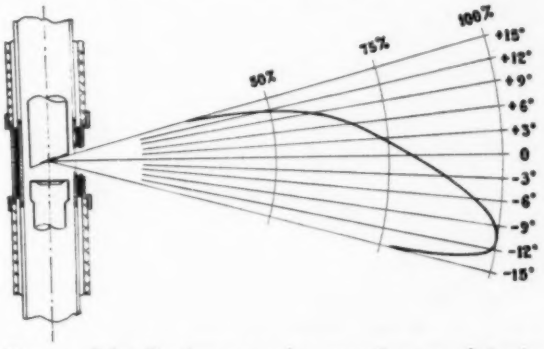

Fig. 1. Distribution on polar coordinates of the intensity at uniform distance of unfiltered radiation at various angles to the central ray from an autoprotective line-focus roentgen tube.

Careful measurements were made at 1° intervals to determine accurately the maximum direction. This was found to occur at −10° when data were taken at 25 cm. central ray distance and at −11° taken at 50 cm. central ray distance. The agreement of the angle of maximum intensity as found at 50 cm. with that at 25 cm. would indicate that there was not sufficient scattering from the few exposed parts of the tube holder to materially affect the results. The rapid decrease in intensity at greater negative angles is undoubtedly due to the eclipse of the focal area by the exit portal. Similar measurements on the non-protective Media tube showed a maximum at −12°, a good correlation with the Metalix. This would seem to indicate that on the line-focus tubes with the anode face at 70° to the tube axis, the intensity of radiation increases with increasing angle from the target face through about 30° to 35°, which is in accord with the results obtained by Coolidge and Kearsley on a 45° target.

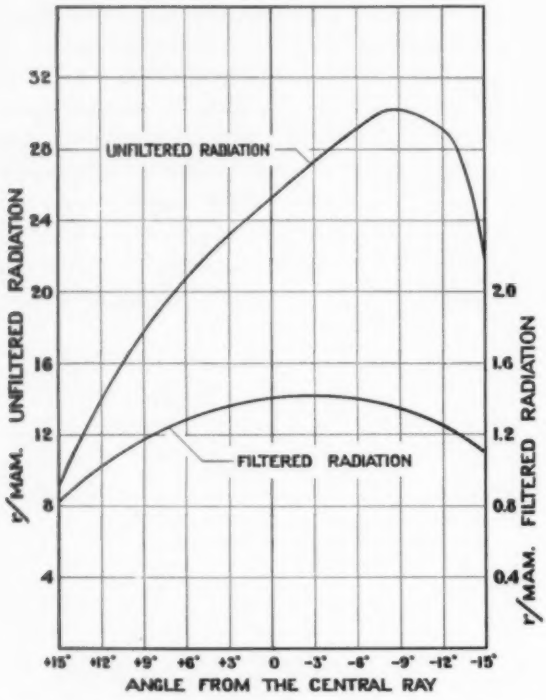

Fig. 2. Plot on rectangular coordinates of the intensity on a plane parallel to the tube axis, at various angles to the central ray, of unfiltered radiation and of radiation filtered by approximately 5 mm. of aluminum, from an auto-protective line-focus roentgen tube.

When the above data are reduced to intensity on a plane parallel to the tube axis the maximum is found at $-8°$ to $-9°$, as is shown in Figure 2. Figure 2 also shows the intensity on a plane from the Metalix tube after passing through a filter of approximately 5 mm. of aluminum positioned in the filter slots of the tube holder. This is more representative of the conditions met in practical roentgenography since the body of the patient absorbs radiation equivalent nearly always to at least this much. It will be noted that the maximum is here nearly at the central ray and that the decrease on either side is not as marked as without filtration. Although under the arrangement used a part of the decrease on either side of the central ray is due to the increased thickness of aluminum traversed at the larger angles, it is evident that there is definitely less radiation near the periphery of the emergent cone, especially toward the anode end where at $+15°$ the intensity is only about half that of the central ray.

The Quality at Various Angles to the Central Ray. Differences in the quality of beams of heterogeneous radiation are best shown by the relative absorption curves;[5] data for these from the Metalix tube in four directions are tabulated in Table 1. Here, too, the intensity readings of unfiltered radiation were in error due to the wall effect of the thimble chamber, but the quality of radiation at the voltage used on readings taken with more than 0.5 mm. of aluminum and up to over 13 mm. of aluminum was such that the correction factor for the roentgenometer used was less than its rated 2 per cent accuracy. Therefore, it was deemed better to base the curves upon an initial filtration of 1 mm. of aluminum as the 100 per cent value. The intensities through exactly 1 mm. of aluminum were not obtainable by direct reading due to the fact that the filter thickness traversed in-

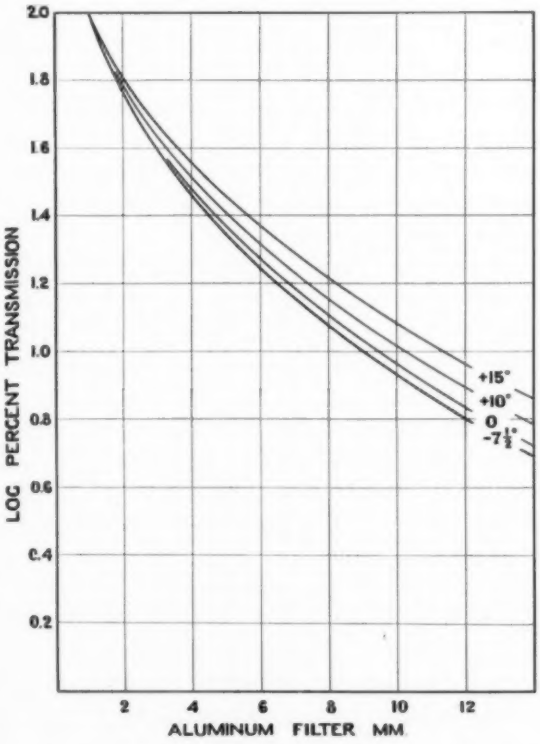

Fig. 3. Relative aluminum transmission curves of the radiation at various angles to the central ray from a line-focus roentgen tube.

creased with the angle to the central ray, so these figures were obtained by plotting an absorption curve using the zero filtration as 100 per cent and interpolating the intensity at exactly 1 mm. Curves based upon the interpolated value of 1 mm. of aluminum as the 100 per cent intensity are plotted in Figure 3. These show that the radiation becomes progressively harder from the direction of maximum intensity toward the projection of the anode face.

TABLE I

TRANSMISSION THROUGH ALUMINUM OF RADIATION FROM A LINE-FOCUS ROENTGEN TUBE (METALIX) AT DIFFERENT ANGLES TO THE CENTRAL RAY

$-7\frac{1}{2}°$

Filter	r/ma-min.	Per cent	Per cent
0	28.2	100	—
1.00	6.96*	24.7	100
1.06	6.78	25.05	97.2
2.02	4.01	14.21	57.5
3.29	2.54	9.02	36.5
4.06	2.00	7.11	28.8
5.31	1.45	5.15	20.8
6.11	1.21	4.30	17.4
8.16	0.809	2.87	11.6
10.19	0.579	2.05	8.30
13.49	0.367	1.30	5.26

0°

Filter	r/ma-min.	Per cent	Per cent
0	24.65	100	—
1.00	6.74*	27.3	100
1.05	6.55	26.6	97.2
2.00	3.94	15.97	58.5
3.26	2.53	10.25	37.6
4.03	2.00	8.11	29.7
5.26	1.50	6.09	22.3
6.07	1.25	5.07	18.55
8.10	0.839	3.40	12.45
10.10	0.610	2.47	9.06
13.36	0.387	1.57	5.74

+10°

Filter	r/ma-min.	Per cent	Per cent
0	17.4	100	—
1.00	5.10*	29.3	100
1.066	4.90	28.1	96.2
2.03	3.09	17.75	60.7
3.31	2.03	11.62	39.8
4.09	1.61	9.26	31.6
5.34	1.21	6.95	23.8
6.15	1.01	5.77	19.74
8.21	0.703	4.04	13.80
10.25	0.505	2.90	9.92
13.56	0.326	1.87	6.40

+15°

Filter	r/ma-min.	Per cent	Per cent
0	10.6	100	—
1.00	3.56*	33.7	100
1.09	3.39	32.0	95.2
2.10	2.20	20.80	61.7
3.38	1.47	13.90	41.3
4.17	1.24	11.70	34.8
5.47	0.940	8.87	26.7
6.28	0.797	7.53	22.4
8.39	0.553	5.22	15.50
10.46	0.401	3.83	11.27
13.81	0.266	2.52	7.46

* Value derived from curve.

Discussion. The curve of Figure 1 indicates that there is a marked difference in the output of unfiltered radiation at various angles to the central ray on line-focus tubes and the absorption curves of Figure 3 indicates that the quality of radiation likewise varies considerably. For this reason, in any researches in which the output of a line-focus roentgen tube may be a factor, care must be taken to select and use a beam in only one particular direction. Slight variations in the positioning of the tube which give differences in the incident direction may lead to discordant results especially in experiments where little or no filtration is used. Care should also be taken that the experiment be made at such a distance from the focus that only a small solid angle of radiation is intercepted, lest both the quantity and quality over its area vary too greatly from uniformity.

In practical roentgenography also these variations are of definite significance although as shown by the curve of Figure 2,

Table II

TRANSMISSION THROUGH ALUMINUM OF THE CENTRAL RAY FROM A MEDIA ROENTGEN TUBE

Filter	r/ma-min.	Per cent	Per cent
0	15.82	100	—
0.50	7.75	49.0	—
1.00	5.34*	33.7	100
1.05	5.18	32.7	97.1
2.00	3.12	19.7	58.5
4.03	1.54	9.76	28.9
5.26	1.13	7.14	21.1
6.06	0.925	5.85	17.3
8.09	0.622	3.93	11.7
10.10	0.434	2.74	8.14
13.36	0.263	1.66	4.94

* Interpolated value.

where intensities were measured after passing through 5 mm. of aluminum, the variation from the maximum at angles distant to the central ray is not so marked. For instance, abdominal films of 14″×17″ size exposed from a line-focus tube at a target distance of 25 to 28 inches show a definite weakening of exposure on the end in the direction of the anode. If such films are made with the anode cephalad, the loss of the exposure may impair the diagnostic value of such films as one of the kidney, ureter and bladder or of the spine; but in exposures with the anode caudad, the lesser intensity is directed toward the pelvic region which is usually not as thick as the upper abdomen and a better roentgenogram results. Where the radius of the Potter-Bucky will permit, a distance of not less than 30 inches should be used. Again, in chest roentgenography an abundance of soft radiation is usually desired toward the apex, which is not as highly ventilated as the lower lobes; this may be partially realized by positioning the tube so that the anode is down and radiation at angles toward the cathode is directed upward.

II. OUTPUT AS AFFECTED BY THE CHARACTERISTICS OF THE ROENTGEN TUBE AND THE ROENTGEN MACHINE

Difference in the Output of Different Tubes. Using the same applied voltage as indicated by the prereading meter on the control panel, data for the aluminum absorption curve of the central ray from the Media tube were obtained and are presented in Table II. These data were reduced to a basis of 1 mm. as the 100 per cent value as described above, and the relative transmission curve so obtained is plotted in Figure 4 along with that of the central ray from the Metalix tube. In spite of the fact that the same primary voltage was applied to the high tension system, these data indicate a definite difference in both the quality and quantity output of the two tubes. The Media tube has a small diameter glass bulb for the discharge chamber and is not auto-protective as compared with the chrome-iron cylindrical discharge chamber of the Metalix type tube. Thus it seems possible that the characteristics of the roentgen tubes may have a definite effect upon the high tension, although these differences may also be due, at least

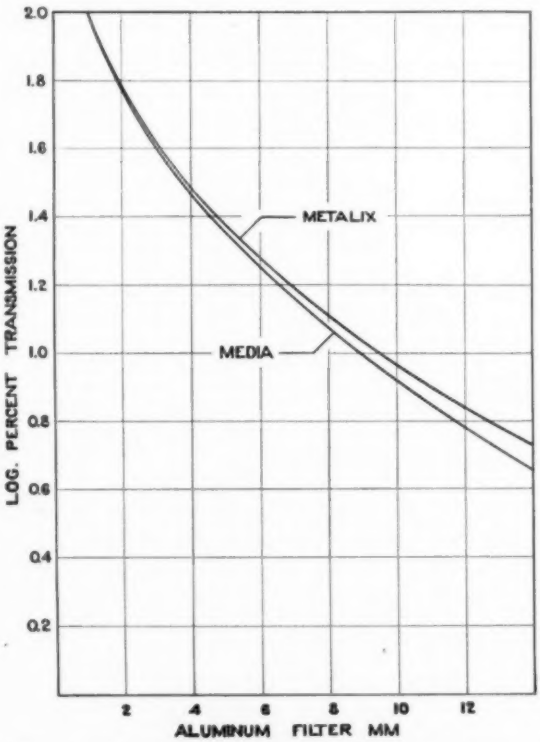

Fig. 4. Relative aluminum transmission curves of the central ray from a non-protective roentgen tube (Media) compared with that from a tube with a chrome-iron discharge chamber (Metalix).

in part, to the thickness and composition of the glass envelopes.

Output as Affected by the High Tension Generator. Some nine months after the previous data were obtained all four valve tubes of the transformer rectifying system had burned out and had been replaced by some of a different type. To determine what, if any, effect this might have on the output of the roentgen tube, an absorption curve was taken of the central ray of the Metalix tube under the same conditions of control setting previously used. The data are presented in Table III and the relative transmission curve in comparison with the older run is plotted in Figure 5. These show a small but definite change in both the quantity and quality of the output.

Discussion. In practical roentgenography, regardless of whether the equipment may have been calibrated by sphere gap measurements or by some other method, the exposure made is always based upon the primary controls either through setting the auto-transformer to a definite "button" or to a definite meter reading of primary voltage (although the latter may read secondary kilovoltage). The relative transmission curves of Figure 4 indicate that even though the same primary voltage be applied to the generator, different (type) roentgen tubes may give an output which is different in both intensity and quality. The data of Table III and Figure 5 indicate that a change in the characteristics of the high tension generator may likewise affect the roentgen output both as to quality and quantity. For this reason, calibration, especially for research work, to be exact, should be made for each tube used and should be repeated whenever any change is made in either the roentgen tube or the rectifying system.

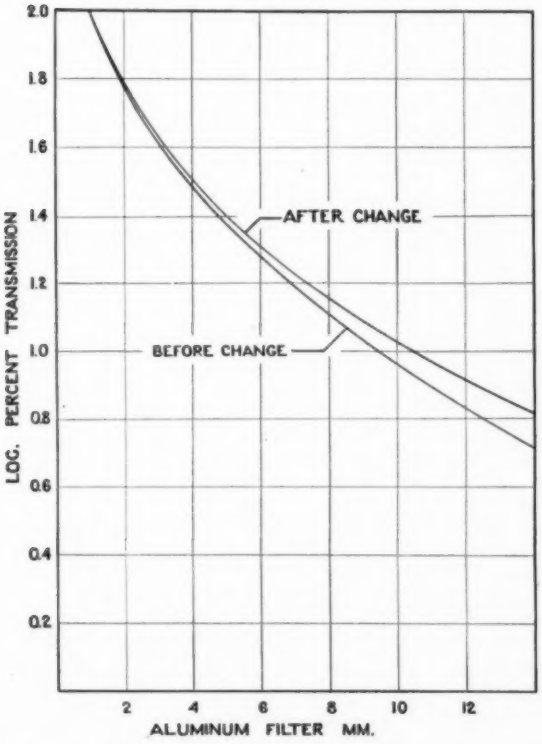

Fig. 5. Relative aluminum transmission curves of the central ray from the same roentgen tube before and after change of the generator rectifying tubes to those of a different type.

Table III

TRANSMISSION THROUGH ALUMINUM OF THE CENTRAL RAY FROM THE METALIX TUBE AFTER A CHANGE IN THE RECTIFYING TUBES OF THE GENERATOR

Filter	r/ma-min.	Per cent	Per cent
0	28.9	100	—
1.00	6.07*	21.0	100
1.05	5.91	20.4	97.4
2.00	3.68	12.7	60.5
3.05	2.50	8.65	41.1
4.03	1.87	6.49	30.9
5.08	1.50	5.19	24.7
6.06	1.21	4.20	20.0
8.09	0.854	2.95	14.0
10.09	0.644	2.22	10.6
13.35	0.427	1.48	7.05

* Interpolated value.

SUMMARY

An experiment is described in which ionization measurements were made of the intensity in the plane of the central ray and normal to the anode face, of the output from a line-focus roentgen tube at various angles to the central ray. These showed that the unfiltered radiation varied quite markedly at different angles, especially toward the anode face, and that the maxi-

mum intensity was to be found at about 10° from the central ray toward the cathode in the tube used. The intensity of radiation filtered through 5 mm. of aluminum showed much smaller variation but was considerably less than that of the central ray at angles of 10° to 15° away. Aluminum transmission curves taken at different angles revealed that the quality of the radiation likewise varied, showing a progressive hardening from the direction of maximum intensity toward the projection of the anode face.

A relative transmission curve of the central ray from a non-protective line-focus roentgen tube when compared with that from an auto-protective tube with a chrome-iron discharge chamber taken under the same conditions of applied primary voltage showed an output of radiation that was somewhat different both as to quality and quantity.

Comparison of the relative transmission curves from the same roentgen tube taken after a change of the rectifying tubes to those of another type revealed that the output of radiation was different both as to quality and quantity.

These results may be of considerable significance in researches involving radiation of the diagnostic range and suggest the necessity of definite precautions.

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EDITORIALS

WILLIS FASTNACHT MANGES
1876–1936

IN THE full flower of a life of exceptional vocational activity, a life continued from day to day in a spirit of joyous enthusiasm that the passing years could not quench, whether evinced in earnest scientific pursuit or in lighter moments of recreational relaxation, Dr. Willis Manges, on the twenty-fourth day of November, 1936, was suddenly lost to his colleagues of the profession on which he had for so long shed luster, and to humanity in affliction on behalf of which he had labored so beneficently.

Doctor Manges was born in 1876 at Luthersburg, Pennsylvania, a son of the Reverend Edmund and Margaret (Whisker) Manges. On the completion of his preliminary education at Gettysburg Academy and Gettysburg College, he entered Jefferson Medical College in Philadelphia where, through assiduous self-help and with scholastic brilliance, he was graduated in 1903.

With a mind of mechanical turn and with earnest intent in matters pertaining to physics, he had become, long before the day of his degree in Medicine, so completely fascinated by the rapidly expanding revelations of the roentgen rays that he resolved to make the theory and practice of the "new science" the basis of his professional endeavor. In consequence he embraced a year's special training in "x-ray work" under Drs. Sterrett Metheny and William M. Sweet during the late course of his interneship at the Jefferson Hospital. In June, 1904, at the end of his term as House Officer, Doctor Manges was appointed Roentgenologist to succeed Doctor Metheny, who desired to discontinue the work. In 1925, he was made Clinical Professor of Roentgenology at Jefferson Medical College and, in due course (1929), Professor. For over thirty-two years he had been chief of his hospital department.

Concerning his early days at the Jefferson Hospital, Doctor Manges later referred to the year 1907 as marking his acquisition of the very first generator of the transformer type with rotary rectification to be built by Dr. H. C. Snook, its inventor. This historical piece of apparatus was in daily use in his Department for twenty-five years from the day when its installation terminated the era of the static machine and the induction-coil with its interrupter.

It was in 1907 also that Willis Manges became a member of the American Roentgen Ray Society. It was but four years later that he was elected one of its officers in recognition of his sterling contribution, made in 1910, on the subject of female pelvic measurement. From then on his participation in the affairs of the Society was active and loyal. He became Secretary in 1914 and held that office for three years; he was elected President for 1918, the year of his remarkable military service; he was made a member of the Executive Council in 1922 and 1923, and its Chairman in 1924. In 1929, he again became a member of the Council and was again elected its Chairman in 1930. This record is incontrovertible evidence of the confidence placed in Manges by his professional colleagues, who early recognized the fiber of his character and the quality of his executive ability in matters involving the welfare of the Society. It goes without saying that Doctor

WILLIS FASTNACHT MANGES
1876–1936

UNIV. OF MICH.

Manges took equally keen interest in his local scientific societies and in roentgenological organizations other than the older national association. He was prompt to affiliate with that group of scientists called together in 1906 by Charles Lester Leonard to form the Philadelphia Roentgen Ray Society, and had been intimately identified with its affairs for years. He gave energetic aid to the finally successful organization of a Section on Radiology of the American Medical Association in 1924. He was intensely jealous of the sound ethics and good name of his specialty, and toward their maintenance gave fully of his time and labor in the establishment of the American Board of Radiology, of which he was one of the original members.

Doctor Manges was seriously appreciative of his affiliation with medical organizations of wider geographical distribution, both within his specialty and outside it, and in return accorded them generous cooperation when occasion and his judgment required. He was a member of the Pennsylvania State and Philadelphia County Medical Societies, and a Fellow of the College of Physicians of Philadelphia, the American College of Physicians and the American College of Radiology.

From the day, in the autumn of 1910, of his memorable paper, already mentioned, before the American Roentgen Ray Society, Manges' contributions to the many phases of radiologic practice were of such sound quality and so definitely indicative of his versatility that his colleagues of the past three decades now living may well find it difficult consonantly to appraise those of his works which remain most conspicuously monumental of his career. It is probable that perfect agreement would be reached on at least two. One of these is his notable military service during the World War as medical officer in charge of the Camp Greenleaf School of Roentgenology at Fort Oglethorpe, Georgia. During the year following the termination of the War, Colonel Manges described in great detail, in the American Journal of Roentgenology, the specific activities of this School and the machinery of its organization, but in that communication and elsewhere with characteristic modesty he hides the light of his own efficient directorship and its results under the bushel of his praise of the coordinating work of his fellow officers of the executive staff of the School. After the School became functionally active through the efforts of Manges and his co-organizers, the effect of their combined patient instruction was soon to be seen in the caliber of the Medical Corps personnel, of all ranks, whom they had trained for military roentgenological work in various capacities. But, as History records in connection with more than one branch of our huge military preparation and, as Colonel Manges himself wrote later, " . . . the Armistice came before the full benefit of the instruction and equipment was felt in the War zone."

As another immovable monument to Manges' career, one ventures to indicate his long and persistent labor, in association with certain of his clinical colleagues, in furtherance of the efficacious application of the roentgen rays to the localization and removal of foreign bodies, radiolucent as well as radiopaque, in the respiratory passages, the eye and the upper alimentary tract. The manipulative skill of these men, coordinating with that of one to whom a rare power of fluoroscopic discernment came almost as a supernal gift, established the prototype of perfect surgical, bronchoscopic and roentgenoscopic alliances that have restored thousands to normal well-being.

Blessed with an ever increasing amount of clinical material that only such ideal coadjuvancy could assure, Manges wrote repeatedly on the subject of this type of roentgenological practice for fully fifteen years, the subject of non-opaque foreign bodies having been masterfully presented by him at the First International Congress of Radiology at London in 1925. Very recently, Manges reviewed his entire experience in this special field in his Carman Lecture for 1936.

For as many years Doctor Manges strove constantly, and with historical success, to perfect the technique of foreign-body localization in regions of anatomic complexity. He soon embraced the use of the biplane roentgenoscope, after observing its results in the pioneer hands of Grier of Pittsburgh. Manges applied this apparatus with great success to non-magnetic foreign bodies in the eye, especially the vitreous.

The constructive effect of Manges' creative instinct was shown early in other fields of roentgenological practice. His first communication (1910) to the American Roentgen Ray Society dealt with a method of measuring the female pelvis which involved the combined employment of stereoroentgenography and the Mackenzie-Davidson crossed-threads principle of localization. Manges' procedure did not demand so much of the patient by way of difficult posture in relation to the plate (film) and its value, established at the time, accords to it a distinct position among the many pelvimetric methods that progress has evolved.

Upon resumption of civil practice after the War, Doctor Manges directed much of his investigation to further observation of intrathoracic change—change in lung, diaphragmatic function, heart and mediastinal contents consequential to the presence of inspired foreign bodies, and to the study of the esophagus, its anomalies and mural abnormalities, throughout the course of which his ideal relationship with his surgical coworkers was maintained. He became much interested in nasal accessory sinus infection and its relation to respiratory intrathoracic disease, including asthma, and wrote frequently on this subject. His routine paranasal sinus investigation was carried out for years with an apparatus of his own devising which allowed of the upright position of the head, with air-compression of screen and film.

His experience with fluoroscopy even at that day made of Manges an early advocate of roentgen pyeloscopy. Undertaking this procedure as early as 1912, his original purpose in adopting it was to control overdistention of the renal pelvis by radiopaque delineating solutions, but it was later "always done, not only for the urologist, but for the surgeon and gynecologist . . . [and] . . . indicated in every case and not only in the exceptional or selected case."

During the latter years of his life, Doctor Manges enjoyed consistent success in the roentgen therapy of acute and subacute infections, applying small doses. His discussions of his results, undertaken on several occasions were, as usual, most temperate in vein, and were supported by the citation of many conclusive case reports.

The girders which supported the structure of Willis Manges' character as a physician were professional honesty, generosity, kindliness, determination. He was honest with himself, with his colleagues and with his patients. He permitted no self-delusions, therefore it was spiritually impossible for him to disseminate delusion where others were concerned. He loved the truth because it was the truth; he abhorred sham, indirection and all sinister dealings masquerading under the guise of science. His generosity, especially toward his fellow practitioners, is shown by a perusal of almost any of his writings dealing with matters wherein his coworkers were associated; these are usually prefaced by words of credit to them and of dignified depreciation of his own participation. His loving kindness is revealed repeatedly in the pains he took to augment the comfort of his patients. Most of his work was of such nature that it could be pursued successfully only by a lover of children both young and old —for the adult in physical distress is once again but a little child—who, if need there were, came to him the second time gladly and with no fear in their hearts. His determination, his high purpose, was evident in everything he undertook, whether it were the slight task of the day or the leadership of a great War training school.

Willis Manges possessed a sense of humor of a quality that could not but have relieved his moments of fatigue, discour-

agement and vexation, even though at times it perforce concealed the anguish of the sympathetic heart of this true physician. For most of us it was irrepressible—flashing out even in his scientific writings, in one of which, for example, he observed, in considering the uses to which roentgen rays are today applied in the industrial arts and with his beloved pastime in mind: "Golf balls are now being x-rayed to determine the size, the symmetry and the correct position of the core. (It should be understood, however, that this is being done by the manufacturer and not by medical roentgenologists who happen to play golf.)"

A devoted lover of wholesome sport for its own sake, to the annual meetings of his colleagues in Radiology Willis Manges often came early for the usual pre-sessional enjoyment of outdoor activity, his joyous laugh portending his approach, the implements symbolic of his athletic prowess swinging from his shoulder as happily as ever the mythological *Eros* bore his quiver of arrows, bringing the fresh air of vivacity to blow the pungent atmosphere of fluoroscopy from our lungs, the dust of libraries from our eyes and to clarify our brains for the more momentous work of our assembly.

* * * * *

American Roentgenology has lost another ornament from the diadem she wears so proudly. How readily and how truly we can say of Willis Manges, as but last year he himself, in the Carman Lecture, said of Russell Carman, quoting the words that the late Jacob Mendez Da Costa used in eulogy of his eminent contemporary, Samuel D. Gross: "He died too soon, for he was in fine possession of his splendid powers and was doing his best work!"

Percy Brown

RESOLUTION OF THE PHILADELPHIA ROENTGEN RAY SOCIETY ON THE DEATH OF WILLIS FASTNACHT MANGES

Whereas, our beloved fellow member, Willis Fastnacht Manges, was taken from us by death on November 24, 1936, and

Whereas, we fully appreciate the magnitude of our loss, and desire to express and permanently record our thoughts upon this sad occasion, be it therefore

Resolved, that the following statement be spread upon our minutes and a copy sent to the bereaved family of our departed friend.

Willis Manges combined, in rare degree, the qualities of Scientist, Teacher, Physician and Gentleman. Born at Luthersburg, Pennsylvania, in 1876, he attended Gettysburg College, then matriculated at Jefferson Medical College and was granted the degree of Doctor of Medicine in 1903. Twenty-five years later (1928) Gettysburg recognized his preëminence by bestowing upon him the Honorary Degree of Doctor of Science.

He began the practice of Radiology when that specialty was still in its infancy. He can therefore be listed as one of Radiology's Pioneers. From the very beginning of his work he gave evidence of unusual ability along investigative lines. Never content merely to follow the beaten path, he was often the first to try out a new technique or method. Radiologists all over the world are aware of their debt to him, for he not only accomplished fundamental researches but he also enriched the scientific literature with many a lucid description of his results. To mention only the more outstanding of his contributions, we call attention to (1) his announcement of the first mathematically accurate method of roentgen pelvimetry, published in the *American Quarterly of Roentgenology* for 1911; (2) his introduction of pyeloscopy as an adjunct and control for pyelography, first reported in the American Journal of Roentgenology for 1918; (3) his development of

an efficient organization at Camp Greenleaf where hundreds of Army Medical Officers were given training in Roentgenology under his direction during the World War; (4) his vivid description of the mechanism and diagnostic significance of obstructive emphysema and atelectasis, in connection with foreign bodies and various endobronchial lesions; (5) his work with Chevalier Jackson in the application of biplane roentgenoscopy to the problem of recovering foreign bodies from the air and food passages; (6) his successful application of the biplane roentgenoscope to the problem of localizing and removing non-magnetic foreign bodies from the eye.

As a teacher, Willis Manges was gentle, lucid, painstaking, and inspiring. Fortunate indeed are those who can say, "I was his understudy." And fortunate was his Alma Mater in her choice of Willis Manges as Professor of Roentgenology.

Willis Manges was first and foremost a physician. He realized from the beginning of his work in roentgenology that the importance of developing technique must not be permitted to turn the physician-roentgenologist into a technician. He warned us, frequently, that we must be physicians first, roentgenologists second. As a Charter Member of the American Board of Radiology his voice was raised in defense of proper standards in this matter.

Finally, we pay tribute to Willis Manges, the Man. The quality of his friendship warmed our hearts. His personal charm will never be forgotten. His generosity and unselfishness made the world a better place for us to live in. He set an example to all in his integrity and character. His passing has made us poorer, as his years with us made us richer. We glory in his achievements and will ever cherish the memory of his years in our company.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. E. P. Pendergrass, University Hospital, Philadelphia, Pa. Annual Meeting: Chicago, Ill., Sept. 13–17, 1937.

American College of Radiology
Secretary, Dr. B. H. Orndoff, 2561 N. Clark Street, Chicago, Ill. Annual meeting: Atlantic City, N. J., June, 1937.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Atlantic City, N. J., June 9–11, 1937.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N.Y. Annual meeting: Chicago, Ill., Sept. 13–17, 1937.

Radiological Section, Baltimore City Medical Society
Secretary, Dr. Howard H. Ashbury, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut State Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets annually in May. Special meetings may be called by the Chairman.

Radiological Section, Los Angeles County Medical Society
Secretary, E. N. Liljedahl, Los Angeles, Calif. Meets on the second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. E. Mendelson, 120 E. 19th St., Brooklyn, N. Y. Meets monthly on first Tuesday, October to April.

Buffalo Radiological Society
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

Chicago Roentgen Society
Secretary, Dr. R. G. Willy, 1138 N. Leavitt. Meets second Thursday of each month October to May inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. H. G. Reineke, General Hospital Cincinnati, Ohio. Meets third Monday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. North W. Shetter, 14600 Detroit Ave. Meetings are held at 6:30 P.M. at the Cleveland Chamber of Commerce Club rooms on the fourth Monday of each month from October to April, inclusive.

Denver Radiological Club
Secretary, Dr. K. D. A. Allen, 452 Metropolitan Building, Denver, Colorado. Meets on any month having a fifth Monday.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne Country Medical Society Building.

Florida State Radiological Society
Secretary, Dr. Harold O. Brown, Tampa, Florida. Meetings held twice a year, May and November.

Illinois Radiological Society
Secretary, Dr. Cesare Gianturco, 602 W. University Ave., Urbana, Ill. Regular meetings held quarterly.

Indiana Roentgen Society
Secretary, Dr. C. C. Taylor, Indianapolis, Ind. Meeting held the second Sunday in May annually.

Long Island Radiological Society
Secretary, Dr. S. G. Schenck, 115 Eastern Parkway, Brooklyn, N.Y. Meets at Kings County Med. Soc. Bldg. on third Thursday of each month from October to May, at 8:30 P.M.

Michigan Association of Roentgenologists
Secretary, Dr. C. S. Davenport, St. Lawrence Hospital, Lansing, Mich. Three meetings a year, Fall, Winter, and Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. S. A. Morton, Columbia Hospital, Milwaukee, Wis. Meets monthly. Place of meeting named by the president.

Minnesota Radiological Society
Secretary, Dr. L. G. Rigler, University Hospital, Minneapolis, Minn.

Nebraska State Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday evening of each month.

New England Roentgen Ray Society
Secretary, Dr. E. C. Vogt, 300 Longwood Ave., Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. P. S. Avery, New Brunswick, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place designated by the president.

New York Roentgen Society
Secretary, Dr. R. W. Lewis, 140 E. 54th St., New York. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse, N. Y. Three meetings a year—January, May, and November.

Pacific Roentgen Club
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, Williamsport, Pa. Two day annual meeting held in May or June. Time and place of next meeting to be announced later.

Philadelphia Roentgen Ray Society
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Camp C. Thomas, 476 Lake Ave. Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

St. Louis Roentgen Ray Society
Secretary, Dr. P. C. Schnoebelen, Missouri Bldg., St. Louis, Mo. Meets first week of each month. Time and place of meetings designated by president.

South Carolina X-Ray Society
Secretary, Dr. R. B. Taft, 105 Rutledge Ave., Charleston. Meets at time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. J. H. Smith, Lubbock, Texas
Meets annually one day preceding the meeting of the Texas State Medical Association.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Secretary, Dr. C. B. Peirce, University Hospital, Ann Arbor, Mich. Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

VIRGINIA ROENTGEN RAY CLUB
Secretary, Dr. V. W. Archer, University Hospital, University, Va.
Meets annually in October.

CUBA

SOCIEDAD CUBANA DE RADIOLOGIA Y FISIOTERAPIA
Secretary, Dr. Francisco Padron, Enrique, Villuendas 64, Havana, Cuba. Meets monthly in Havana.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Meets on the third Thursday of each month, from November to June inclusive, at 8:15 P.M., at 32 Welbeck St., London, W. 1, or as advertised.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to Nov. incl. for scientific discussions.

SECTION ON RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. A. H. Rolph, 160 St. George St., Toronto, Ont.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION
Secretary, Dr. P. C. Fenwick, The Hospital, Christchurch. Meets annually.

CONTINENTAL EUROPE

BELGIAN SOCIETY OF ROENTGENOLOGY
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

SOCIEDAD ESPAÑOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ DE RADIOLOGIE MÉDICALE DE FRANCE
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel.
Meets annually in different cities.

SOCIÉTÉ FRANCAISE D'ELECTROTHÉRAPIE ET DE RADIOLOGIE MÉDICALE
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

ASSOCIATION OF GERMAN ROENTGENOLOGISTS AND RADIOLOGISTS IN CZECHO-SLOVAKIA
Secretary, Dr. Walter Altschul, German University, Prague, 11.52.

DEUTSCHE RÖNTGEN-GESELLSCHAFT (GESELLSCHAFT FÜR RÖNTGENKUNDE UND STRAHLENFORSCHUNG)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

SÜD- UND WESTDEUTSCHE RÖNTGENGESELLSCHAFT
Meets annually in different cities.

NORD- UND OSTDEUTSCHE RÖNTGENGESELLSCHAFT
Meets annually in different cities.

DUTCH SOCIETY OF ELECTROLOGY AND ROENTGENOLOGY
Holds two meetings a year in Amsterdam, one in the Spring, and one in the Fall.

SOCIETA ITALIANA RADIOLOGIA MEDICA
Secretary, Professor M. Ponzio, University of Turin, Turin.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. G. Gatoschi, Str. Isvor 14–16, Bucarest.
Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson.
Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gusterin.
Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.
Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

POLISH SOCIETY OF RADIOLOGY
Secretary, Dr. Jan. Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

WARSAW SECTION, POLISH SOCIETY OF RADIOLOGY
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime.

SOCIETY OF MEDICAL RADIOLOGY IN SWEDEN
Meets in Stockholm.

SOCIETY OF MEDICAL RADIOLOGY IN NORWAY
Meets in Oslo.

SOCIETY OF MEDICAL RADIOLOGY IN DENMARK
Secretary, Dr. G. Biering, Copenhagen.
Meets on the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

SOCIETY OF MEDICAL RADIOLOGY IN FINLAND
Meets in Helsingfors.

VIENNA ROENTGEN SOCIETY
Meets first Wednesday of each month, at 6.30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

ORIENT

JAPAN X-RAY ASSOCIATION
c/o Orthopedic Surgery, Tokyo Imperial University.
Meets annually in April.

KINKI ROENTGEN-ABEND SOCIETY
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto Japan. Meets bi-monthly on third Sunday.

THE PROGRAM OF THE FIFTH INTERNATIONAL CONGRESS OF RADIOLOGY

Contributions for the program of the Fifth International Congress of Radiology are now being received. The Congress will be held at the Palmer House, Chicago, September 13–17, 1937.

The rules of the International Congress require that participants in the program be members of the Congress. Announcements setting forth the requirements for membership have been mailed to all members of radiological societies in all countries.

All who wish to take part in the program should apply to the President, Dr. A. C. Christie, 1835 Eye Street, N. W., Washington, D. C., stating the subject which they wish to present and at the same time should send their application for membership in the Congress to The Secretary, Fifth International Congress of Radiology, 2561 North Clark Street, Chicago, Illinois.

If possible, the application for place on the program should be accompanied by a five hundred word abstract and in any case such abstract must be received by the President not later than April 1, 1937, and the full text of the paper not later than May 1, 1937. The abstracts will be published in the three official languages of the Congress at least six weeks prior to the meeting of the Congress. The full text will also be translated into the other two official languages and will be projected on the screen in those languages while it is being delivered.

Prompt transmission of the abstract and full text will greatly facilitate the work of translation, publication of the book of abstracts and preparation of the films for projection.

EASTERN CONFERENCE OF RADIOLOGISTS

The Eastern Conference of Radiologists will hold its annual meeting in New York City on Friday and Saturday, January 29 and 30, 1937. Headquarters will be at the Hotel Pennsylvania.

There will be an informal reception to visiting radiologists and their families on the evening of January 28. The reception will take place at eight o'clock at the Hotel Pennsylvania. Registration will begin on Thursday afternoon, January 28, at the Hotel Pennsylvania.

The sessions of the Conference will be as follows:

Friday, January 29

9.00 A.M.–12.00 M: Memorial Hospital.
1.00 P.M. (promptly): Luncheon at the Presbyterian Hospital.
2.00–4.30 P.M.: Presbyterian Hospital.

Saturday, January 30

9.00 A.M.–12.00 M.: Mt. Sinai Hospital.
1.00 P.M. (promptly): Luncheon New York Academy of Medicine.
2.00–4.30 P.M.: New York Academy of Medicine: Presentation by Lenox Hill Hospital Staff.

Transportation will be furnished between the Hotel and the various meeting places.

CLINICAL CONFERENCE OF MID-WESTERN RADIOLOGISTS

The first annual Clinical Conference of Mid-Western Radiologists is to be held in Rochester, Minnesota, February 12 and 13, 1937. All radiologists are invited to attend. Headquarters will be at the Kahler Hotel.

If there is a sufficient number making the trip from Chicago to Rochester there will be a special train from Chicago on Thursday afternoon, February 11.

SOUTH CAROLINA X-RAY SOCIETY

The first regular fall meeting of the South Carolina X-Ray Society was held on Thursday, November 5, 1936, at 1 P.M. at Charleston in the Medical College of the State of South Carolina. The following program was presented.

1. A Discussion of Supervoltage Therapy Installations. R. B. Taft, M.D.

2. Treatment of Cancer of the Breast. T. Hutson Martin, M.D., and Augusta Willis, M.D.
3. The Effects of Irradiation on Normal and Abnormal Cells. J. Hampton Hoch, D.Sc.
4. Skin Reactions and Skin Recovery. Hillyer Rudisill, Jr., M.D.
5. The Pathological Grading of Tumors. Thomas Peery, M.D.
6. Microscopic Changes in Tumors following Irradiation. Harold Wood, M.D.

The meeting was well attended and it was unanimously voted to make this fall meeting an annual event. As listed in the JOURNAL, the other meeting of the South Carolina Roentgen Society is at the time and place of the State Medical Association meeting. This fall meeting will always be in Charleston on the first Thursday in November which will coincide with the Annual Founder's Day Celebration of the State Medical College.

SILVANUS THOMPSON LECTURE

Dr. Arthur C. Christie, of Washington, D. C., a former Editor of this JOURNAL and President of the Fifth International Congress of Radiology to be held in Chicago in 1937, delivered the Silvanus Thompson Memorial Lecture before the British Institute of Radiology at its Annual Congress in London on December 3, 1936. The subject of his lecture was "The Diagnosis and Treatment of Primary Cancer of the Lungs."

DIPLOMATES OF THE AMERICAN BOARD OF RADIOLOGY

1. ACKEMANN, H. W., Rockford, Ill., *Radiology*.
2. ADAIR, FRANK E., New York, N. Y., *Therapeutic Radiology*.
3. ALBERT, SIMON, Providence, R. I., *Radiology*.
4. ALEXANDER, F. K., Philadelphia, Pa., *Radiology*.
5. ALLEN, B. M., Wilmington, Del., *Diagnostic Roentgenology*.
*6. ALLEN, BUNDY, Tampa, Fla., *Radiology*.
7. ALLEN, KENNETH D. A., Denver, Col., *Radiology*.
8. ALLEN, LEWIS G., Kansas City, Kans., *Radiology*.
9. ALLEN, M. LOWRY, Philadelphia, Pa., *Radiology*.
10. ALLEN, WILLIAM E., JR., St. Louis, Mo., *Roentgenology*.
11. ALLEY, REUBEN G., Pittsburgh, Pa., *Diagnostic Roentgenology*.
12. ALLISON, R. G., Minneapolis, Minn., *Roentgenology*.
13. ALTMAN, W. S., Quincy, Mass., *Radiology*.
14. AMES, FORREST B., Bangor, Me., *Roentgenology*.
15. ANDERSON, W. D., Memphis, Tenn., *Roentgenology*.
16. ANDREWS, J. ROBERT, Cleveland, O., *Radiology*.
17. ANE, J. N., New Orleans, La., *Radiology*.
18. ANSPACH, WILLIAM E., Chicago, Ill., *Roentgenology*.
19. ARCHER, VINCENT W., University, Va., *Roentgenology*.
20. ARENS, ROBERT A., Chicago, Ill., *Radiology*.
21. ARNESON, A. N., St. Louis, Mo., *Therapeutic Radiology*.
22. ASHBURY, HOWARD E., Baltimore, Md., *Radiology*.
23. ATKINS, S. M., Waterbury, Conn., *Roentgenology*.
24. AURELIUS, J. RICHARDS, St. Paul, Minn., *Roentgenology*.
25. AVERY, PHILIP S., New Brunswick, N. J., *Roentgenology*.
26. BACHMAN, M. H., Youngstown, O., *Roentgenology*.
27. BACON, RALPH D., Erie, Pa., *Radiology*.
28. BADER, E. R., Cincinnati, O., *Radiology*.
29. BAILEY, C. O., Los Angeles, Cal., *Therapeutic Radiology*.
30. BAILEY, WILBUR, Pasadena, Cal., *Radiology*.
31. BAKER, CHARLES F., Newark, N. J., *Roentgenology*.
32. BAKER, EDGAR C., Youngstown, O., *Radiology*.
33. BALL, CLARENCE F., Rutland, Vt., *Therapeutic Radiology*.
34. BALL, ROBERT P., Chattanooga, Tenn., *Roentgenology*.
35. BARFIELD-CARTER, M., Birmingham, Ala., *Roentgenology*.
36. BARKER, WALTER C., Philadelphia, Pa., *Radiology*.
37. BARKER, W. ALLEN, Petersburg, Va., *Roentgenology*.
38. BARNES, JOHN M., Buffalo, N. Y., *Roentgenology*.
39. BARNETT, ARTHUR F., Eugene, Ore., *Radiology*.
40. BARR, RICHARD E., Beaumont, Tex., *Therapeutic Radiology*.
41. BARROW, S. C., Shreveport, La., *Radiology*.
42. BARTH, EARL E., Chicago, Ill., *Roentgenology*.
43. BAUER, AUGUST A., Chicago, Ill., *Roentgenology*.
44. BAUM, SAMUEL M., New York, N. Y., *Therapeutic Radiology*.
45. BAXTER, O. D., Sumter, S. C., *Radiology*.
46. BEALS, JOHN A., Greenville, Miss., *Radiology*.
47. BEELER, RAYMOND C., Indianapolis, Ind., *Radiology*.
48. BEILIN, D. S., Chicago, Ill., *Radiology*.
49. BELL, A. L. L., Brooklyn, N. Y., *Roentgenology*.
50. BELL, J. C., Louisville, Ky., *Roentgenology*.
51. BENDICK, ARTHUR J., New York, N. Y., *Radiology*.
*52. BENISHEK, WERNER L., Aurora, Ill., *Roentgenology*.
53. BENJAMIN, EMANUEL W., Providence, R. I., *Radiology*.
54. BERG, H. MILTON, Bismarck, N. D., *Radiology*.
55. BERNATH, GERALD J., Detroit, Mich., *Diagnostic Roentgenology*.
56. BERNSTEIN, J. H., New York, N. Y., *Radiology*.
57. BETHEA, W. R., Memphis, Tenn., *Roentgenology*.
58. BETTS, ARTHUR, Spokane, Wash., *Radiology*.
59. BIRDSALL, EDGAR, Glens Falls, N. Y., *Diagnostic Roentgenology*.
60. BIRKELO, C. C., Detroit, Mich., *Roentgenology*.
61. BISHOP, PAUL A., Philadelphia, Pa., *Radiology*.
62. BLACKETT, CHARLES W., Boston, Mass., *Roentgenology*.
63. BLAINE, EDWARD S., Los Angeles, Cal., *Roentgenology*.
64. BLAKE, T. W., Rochester, Minn., *Radiology*.
65. BLISS, GERALD D., Altoona, Pa., *Radiology*.
66. BLOOM, ARTHUR R., Detroit, Mich., *Diagnostic Roentgenology*.
67. BOGAN, ISABEL K., Brookline, Mass., *Roentgenology*.
68. BOGAN, MARY E., Brookline, Mass., *Roentgenology*.
69. BOGART, FRANKLIN B., Chattanooga, Tenn., *Roentgenology*.
70. BONNAR, JAMES M., New Bedford, Mass., *Radiology*.
71. BONOFF, KARL M., Los Angeles, Cal., *Diagnostic Roentgenology*.
72. BORMAN, C. N., Minneapolis, Minn., *Radiology*.
73. BORZELL, F. F., Philadelphia, Pa., *Radiology*.
74. BOWEN, CARL B., Oakland, Cal., *Roentgenology*.
75. BOWEN, DAVID R., Philadelphia, Pa., *Radiology*.
76. BOWING, HARRY H., Rochester, Minn., *Therapeutic Radiology*.
77. BOYD, JAMES F., Providence, R. I., *Radiology*.
78. BOYES, JAMES G., Plainfield, N. J., *Roentgenology*.
79. BRADLEY, ROBERT A., Atlantic City, N. J., *Radiology*.
80. BRAMS, JULIUS, Chicago, Ill., *Radiology*.
81. BRANDENBURG, H. P., Denver, Col., *Radiology*.

82. Brenneman, R. E., Meadville, Pa., *Radiology*.
83. Bridenbaugh, J. H., Billings, Mont., *Radiology*.
84. Broeser, Henry V., Hoboken, N. J., *Diagnostic Roentgenology*.
85. Bromer, Ralph S., Bryn Mawr, Pa., *Radiology*.
86. Brooksher, W. R., Fort Smith, Ark., *Radiology*.
87. Brouse, Ivan E., Jacksonville, Ill., *Roentgenology*.
88. Brown, H. O., Tampa, Fla., *Roentgenology*.
89. Brown, Percy, Boston, Mass., *Roentgenology*.
90. Brown, Samuel, Cincinnati, O., *Roentgenology*.
91. Bruck, Samuel, Philadelphia, Pa., *Radiology*.
92. Bryan, Lloyd, San Francisco, Cal., *Roentgenology*.
93. Burch, Hobart A., Elmira, N. Y., *Radiology*.
94. Burcham, Thomas A., Des Moines, Ia., *Radiology*.
95. Burnett, H. W., Dayton, O., *Roentgenology*.
96. Butler, Nicholas G., Hartford, Conn., *Radiology*.
97. Butler, P. F., Boston, Mass., *Radiology*.

98. Caldwell, Charles S., Pittsburgh, Pa., *Roentgenology*.
99. Camp, John D., Rochester, Minn., *Roentgenology*.
100. Capp, Charles S., San Antonio, Tex., *Radiology*.
101. Carlson, Glenn D., Dallas, Tex., *Roentgenology*.
102. Carr, Edson W., Chicago, Ill., *Diagnostic Roentgenology*.
103. Carter, Ray A., Los Angeles, Cal., *Roentgenology*.
104. Carty, John R., New York, N. Y., *Radiology*.
105. Case, James T., Chicago, Ill., *Radiology*.
106. Cathcart, John W., El Paso, Tex., *Radiology*.
107. Challenger, Chester J., Chicago, Ill., *Roentgenology*.
108. Chamberlain, W. Edward, Philadelphia, Pa., *Radiology*.
109. Chapman, John F., Pasadena, Cal., *Roentgenology*.
110. Childe, Arthur E., Montreal, Que., *Diagnostic Roentgenology*.
111. Childs, D. S., Syracuse, N. Y., *Roentgenology*.
112. Childs, S. B., Denver, Col., *Roentgenology*.
113. Chilko, A. J., New Rochelle, N. Y., *Roentgenology*.
114. Christie, Arthur C., Washington, D. C., *Radiology*.
115. Cilley, Earl I. L., Bellingham, Wash., *Radiology*.
116. Claiborne, E. M., New York, N. Y., *Radiology*.
117. Clark, D. M., Santa Barbara, Cal., *Radiology*.
118. Clark, James J., Atlanta, Ga., *Roentgenology*.
119. Clark, Stanley A., South Bend, Ind., *Radiology*.
120. Clarkson, Wright, Petersburg, Va., *Radiology*.
121. Cleaves, Edwin N., Boston, Mass., *Diagnostic Roentgenology*.
122. Cleghorn, Charles D., Miami, Fla., *Radiology*.
123. Clement, Gage, Duluth, Minn., *Radiology*.
124. Cleveland, W. R., Evansville, Ind., *Radiology*.
125. Coate, J. D., Oakland, Cal., *Radiology*.
126. Coe, Fred O., Washington, D. C., *Radiology*.
127. Coffin, Whitman K., Boston, Mass., *Roentgenology*.
128. Cohoon, Carl W., Bay Shore, N. Y., *Diagnostic Roentgenology*.
129. Cole, J. M., Windsor, Ont., *Roentgenology*.
130. Cole, Lewis Gregory, New York, N. Y., *Roentgenology*.
131. Cole, Paul F., Springfield, Mo., *Radiology*.
132. Cole, William Gregory, New York, N. Y., *Roentgenology*.
133. Coley, Stephen W., Memphis, Tenn., *Roentgenology*.
134. Collins, E. N., Cleveland, O., *Diagnostic Roentgenology*.
135. Collins, James N., Indianapolis, Ind., *Radiology*.
136. Collins, J. J., Racine, Wis., *Radiology*.
137. Cook, Orrin S., Sacramento, Cal., *Roentgenology*.
138. Cook, Philip H., Worcester, Mass., *Radiology*.
139. Cooley, R. M., Jackson, Mich., *Roentgenology*.
140. Coray, Q. B., Salt Lake City, Ut., *Roentgenology*.
141. Corcoran, William J., Scranton, Pa., *Roentgenology*.
142. Costolow, William E., Los Angeles, Cal., *Therapeutic Radiology*.
143. Crain, C. F., Corpus Christi, Tex., *Radiology*.
144. Cramp, G. W., Brooklyn, N. Y., *Roentgenology*.
145. Crane, A. W., Kalamazoo, Mich., *Roentgenology*.
146. Crawford, Helen L., Winona, Minn., *Roentgenology*.
147. Crosby, Leonard G., Denver, Col., *Radiology*.
148. Crossan, John W., Los Angeles, Cal., *Diagnostic Roentgenology*.
149. Crowder, E. R., Evanston, Ill., *Roentgenology*.
150. Cunningham, Lester W., Mandarin, Fla., *Roentgenology*.
151. Currin, Francis W., Brooklyn, N. Y., *Roentgenology*.
152. Cushway, B. C., Chicago, Ill., *Radiology*.

153. Dann, David S., Kansas City, Mo., *Radiology*.
154. Dannenberg, Max, Brooklyn, N. Y., *Roentgenology*.
155. Danzer, Joseph T., Oil City, Pa., *Radiology*.
156. Dauksys, Joseph, Excelsior Springs, Mo., *Diagnostic Roentgenology*.
157. Davidson, Sol C., Rochester, N. Y., *Roentgenology*.
158. Davis, Ernest L., Springfield, Mass., *Roentgenology*.
159. Davis, F. M., San Antonio, Tex., *Diagnostic Roentgenology*.
160. Davis, H. E., Chicago, Ill., *Radiology*.
161. Davis, Henry B., Lancaster, Pa., *Radiology*.
162. Davis, Kenneth S., Los Angeles, Cal., *Roentgenology*.
163. Davison, C. O., Poughkeepsie, N. Y., *Radiology*.
164. Davison, R. Winthrop, Trenton, N. J., *Roentgenology*.
165. Debbie, Anthony G., St. Albans, N. Y., *Diagnostic Roentgenology*.
166. Decker, Fred H., Peoria, Ill., *Radiology*.
167. Dempster, J. H., Detroit, Mich., *Diagnostic Roentgenology*.
168. Derr, John S., Frederick, Md., *Roentgenology*.
169. Desjardins, Anatole, Wilkes-Barre, Pa., *Roentgenology*.
170. Desjardins, A. U., Rochester, Minn., *Radiology*.
171. Deweese, E. R., Kansas City, Mo., *Radiology*.
172. Dick, Paul G., Chicago, Ill., *Roentgenology*.
173. Dickinson, J. C., Tampa, Fla., *Roentgenology*.
174. Dixon, George S., New York, N. Y., *Diagnostic Roentgenology*.
175. Donaldson, S. W., Ann Arbor, Mich., *Roentgenology*.
176. Doub, Howard P., Detroit, Mich., *Radiology*.
177. Doughty, William M., Cincinnati, O., *Radiology*.
178. Downing, Robert E., Terre Haute, Ind., *Radiology*.
179. Downs, Elwood E., Woodbury, N. J., *Radiology*.
180. Drane, Robert, Savannah, Ga., *Roentgenology*.
181. Dresser, Richard, Boston, Mass., *Radiology*.
182. Duckworth, Roy D., White Plains, N. Y., *Roentgenology*.
183. Duckworth, Willard D., New Rochelle, N. Y., *Roentgenology*.
184. Duffy, James J., New York, N. Y., *Therapeutic Radiology*.
185. Dunham, H. Kennon, Cincinnati, O., *Diagnostic Roentgenology*.
186. Durrance, Fred Y., Houston, Tex., *Roentgenology*.

187. Eastland, William E., Oklahoma City, Okla., *Therapeutic Radiology*.
188. Eastmond, Charles, Brooklyn, N. Y., *Roentgenology*.
189. Edeiken, Louis, Philadelphia, Pa., *Radiology*.
190. Edwards, Harold G. F., Shreveport, La., *Radiology*.
191. Edwards, J. Bennett, Englewood, N. J., *Roentgenology*.
192. Egbert, W. E., Chester, Pa., *Diagnostic Roentgenology*.
193. Ehrenpreis, Bernard, Brooklyn, N. Y., *Roentgenology*.
194. Ehrlich, David E., New York, N. Y., *Radiology*.
195. Eldridge, Watson W., Jr., Washington, D. C., *Roentgenology*.
196. Eley, Clayton W., Norfolk, Va., *Roentgenology*.
197. Ellis, Ivan G., Madison, Wis., *Roentgenology*.
198. Elward, Joseph F., Washington, D. C., *Roentgenology*.
199. Eneboe, John B., San Diego, Cal., *Radiology*.
200. Enfield, Charles D., Louisville, Ky., *Radiology*.
201. Ericksen, Lester G., Dubuque, Ia., *Radiology*.
202. Ernst, Edwin C., St. Louis, Mo., *Radiology*.
203. Erskine, Arthur W., Cedar Rapids, Ia., *Radiology*.
204. Evans, John, Baltimore, Md., *Roentgenology*.
205. Evans, William A., Detroit, Mich., *Radiology*.
206. Eveleth, Fred S., Concord, N. H., *Roentgenology*.
207. Exner, Frederick B., Seattle, Wash., *Radiology*.

208. Farmer, Harry L., Cleveland, O., *Radiology*.
209. Farrell, John T., Jr., Philadelphia, Pa., *Roentgenology*.
210. Faust, J. J., Tyler, Tex., *Radiology*.
211. Feaster, O. O., St. Petersburg, Fla., *Radiology*.
212. Ferguson, James W., Pittsburgh, Pa., *Radiology*.

213. FEUERSTEIN, BENJAMIN L., Bay Shore, N. Y., *Therapeutic Radiology.*
214. FINEMAN, SOLOMON, New York, N. Y., *Roentgenology.*
215. FIROR, WHITMER B., Baltimore, Md., *Roentgenology.*
216. FISHER, J. W., Pittsburgh, Pa., *Radiology.*
217. FISHER, L. F., South Bend, Ind., *Roentgenology.*
218. FLAX, NATHAN, Cincinnati, O., *Roentgenology.*
219. FLINN, F., Decatur, Ill., *Radiology.*
220. FLYNN, JAMES M., Rochester, N. Y., *Radiology.*
221. FOLEY, JOSEPH M., Peoria, Ill., *Roentgenology.*
222. FORD, FRANCES A., Detroit, Mich., *Radiology.*
223. FORT, W. A., Mare Island, Cal., *Roentgenology.*
224. FORTIER, LUCIEN A., New Orleans, La., *Radiology.*
225. FRAY, WALTER W., Rochester, N. Y., *Radiology.*
226. FREEDMAN, EUGENE, Cleveland, O., *Roentgenology.*
227. FREEDMAN, JOHN, Detroit, Mich., *Roentgenology.*
228. FREID, JACOB R., New York, N. Y., *Radiology.*
229. FRERE, JOHN M., Chattanooga, Tenn., *Roentgenology.*
230. FRICKE, ROBERT E., Rochester, Minn., *Therapeutic Radiology.*
231. FRIEDLAND, HENRY, New York, N. Y., *Diagnostic Roentgenology.*
232. FRIEDMAN, ASA B., Brooklyn, N. Y., *Radiology.*
233. FRIEDMAN, HARRY F., Boston, Mass., *Radiology.*
234. FRIEDMAN, JACOB, New York, N. Y., *Roentgenology.*
235. FRIEDMAN, LEWIS J., New York, N. Y., *Diagnostic Roentgenology.*
236. FRIEDMAN, MILTON, Newark, N. J., *Therapeutic Radiology.*
237. FRUCHTER, JOSEPH M., Philadelphia, Pa., *Radiology.*
238. FUGATE, ISAAC T., Louisville, Ky., *Radiology.*
239. FULMER, CHARLES C., San Francisco, Cal., *Roentgenology.*
240. FULTON, HUSTON F., Columbus, O., *Diagnostic Roentgenology.*
241. FUREY, WARREN W., Chicago, Ill., *Radiology.*
242. FURST, NATHAN J., Newark, N. J., *Diagnostic Roentgenology.*

243. GALANTI, CHARLES P., Chicago, Ill., *Roentgenology.*
244. GARLAND, L. H., San Francisco, Cal., *Radiology.*
245. GATELY, TRACY T., New Orleans, La., *Radiology.*
246. GATES, RUSSELL, Minot, N. D., *Roentgenology.*
247. GELEHRTER, JOSEPH, Philadelphia, Pa., *Therapeutic Radiology.*
248. GEMMEL, J. H., Philipsburg, Pa., *Roentgenology.*
249. GEORGE, ARIAL W., Boston, Mass., *Roentgenology.*
250. GERBER, ISAAC, Providence, R. I., *Radiology.*
251. GERSHON-COHEN, JACOB, Philadelphia, Pa., *Radiology.*
252. GEYMAN, MILTON J., Santa Barbara, Cal., *Diagnostic Roentgenology.*
253. GHRIST, DAVID M., Glendale, Cal., *Roentgenology.*
254. GIANTURCO, CESARE, Urbana, Ill., *Roentgenology.*
255. GILES, ROY G., Temple, Tex., *Roentgenology.*
256. GILLIES, CARL L., Cedar Rapids, Ia., *Radiology.*
257. GILMORE, W. M., Stratford, Ont., *Radiology.*
258. GINGOLD, JOSEPH R., Meadville, Pa., *Roentgenology.*
259. GLOVER, M. H., Wichita Falls, Tex., *Radiology.*
260. GOIN, LOWELL S., Los Angeles, Cal., *Roentgenology.*
261. GOLDEN, ROSS, New York, N. Y., *Roentgenology.*
262. GOLDSMITH, MAURICE F., Pittsburgh, Pa., *Roentgenology.*
263. GOODRICH, MURRAY E., Toledo, O., *Radiology.*
264. GOODWIN, PERRY B., Peoria, Ill., *Radiology.*
265. GORFINKELL, JULIUS, Pittsburgh, Pa., *Diagnostic Roentgenology.*
266. GORSLINE, CLARENCE S., Battle Creek, Mich., *Roentgenology.*
267. GRACE, JOSEPH M., Eloise, Mich., *Radiology.*
268. GRADY, HENRY W., Washington, D. C., *Radiology.*
269. GRAHAM, RALPH S., Sacramento, Cal., *Radiology.*
270. GRANGER, AMEDEE, New Orleans, La., *Radiology.*
271. GRAY, HORACE D., Memphis, Tenn., *Radiology.*
272. GREENFIELD, HENRY, Brooklyn, N. Y., *Radiology.*
273. GRIER, GEORGE W., Pittsburgh, Pa., *Radiology.*
274. GRIMM, H. W., Pittsburgh, Pa., *Therapeutic Radiology.*
275. GROESCHEL, L. B., New York, N. Y., *Radiology.*
276. GROH, JEAN A., Cleveland, O., *Roentgenology.*
277. GROOVER, THOMAS A., Washington, D. C., *Radiology.*

278. HABBE, J. EDWIN, Milwaukee, Wis., *Roentgenology.*
279. HACKNEY, URBAN P., Dallas, Tex., *Radiology.*
280. HADLEY, LEE A., Syracuse, N. Y., *Roentgenology.*
281. HALL, E. WALTER, Detroit, Mich., *Radiology.*
282. HALL, WENDELL C., Philadelphia, Pa., *Radiology.*
283. HAMILTON, W. S., San Antonio, Tex., *Diagnostic Roentgenology.*
284. HAMPTON, A. O., Boston, Mass., *Radiology.*
285. HANKINS, JOHN L., Johnson City, Tenn., *Roentgenology.*
286. HANSEN, CYRUS O., Minneapolis, Minn., *Radiology.*
287. HARDING, D. B., Lexington, Ky., *Radiology.*
288. HARDY, CLYDE C., Omaha, Neb., *Diagnostic Roentgenology.*
289. HARE, HUGH F., Boston, Mass., *Radiology.*
290. HARRINGTON, B. D., Tacoma, Wash., *Radiology.*
291. HARRIS, CLARENCE P., Houston, Tex., *Roentgenology.*
292. HARRIS, JOHN H., Harrisburg, Pa., *Radiology.*
293. HARRIS, MILO T., Spokane, Wash., *Radiology.*
294. HARRIS, T. T., Omaha, Neb., *Radiology.*
295. HARRIS, WILLIAM, New York, N. Y., *Therapeutic Radiology.*
296. HARTUNG, ADOLPH, Chicago, Ill., *Roentgenology.*
297. HASLEY, CLYDE K., Detroit, Mich., *Radiology.*
298. HAUSER, HARRY, Cleveland, O., *Radiology.*
299. HAWLEY, SYDNEY J., Danville, Pa., *Roentgenology.*
300. HAWORTH, WALLACE, Portland, Ore., *Radiology.*
301. HAY, PERCY D., JR., Florence, S. C., *Radiology.*
302. HEACOCK, CHARLES H., Memphis, Tenn., *Roentgenology.*
303. HEALY, THOMAS R., Boston, Mass., *Roentgenology.*
304. HEATLEY, JOHN E., Oklahoma City, Okla., *Diagnostic Roentgenology.*
305. HEBERDING, JOHN, Youngstown, O., *Radiology.*
306. HEFKE, HANS W., Milwaukee, Wis., *Radiology.*
307. HENDRICKS, ELLIOTT M., Fort Lauderdale, Fla., *Roentgenology.*
308. HENDRICKSON, ANNA R., Canton, O., *Roentgenology.*
309. HENLE, CARYE-BELLE, Newark, N. J., *Roentgenology.*
310. HENRY, LUCAS S., Syracuse, N. Y., *Roentgenology.*
311. HERPEL, FREDERICK K., West Palm Beach, Fla., *Roentgenology.*
312. HERRMAN, W. G., Asbury Park, N. J., *Radiology.*
313. HESS, GEORGE H., Uniontown, Pa., *Roentgenology.*
314. HEYLMUN, HARRY H., Long Beach, Cal., *Diagnostic Roentgenology.*
315. HILDRETH, R. C., Ann Arbor, Mich., *Radiology.*
316. HILL, HAROLD A., San Francisco, Cal., *Radiology.*
317. HILL, WALTER C., Cleveland, O., *Roentgenology.*
318. HILT, LAWRENCE M., Springfield, Ill., *Roentgenology.*
319. HILTON, JAMES M., Klamath Falls, Ore., *Roentgenology.*
320. HIRSCH, I. SETH, New York, N. Y., *Radiology.*
321. HODGES, FRED J., Ann Arbor, Mich., *Radiology.*
322. HODGES, FRED M., Richmond, Va., *Radiology.*
323. HODGES, PAUL C., Chicago, Ill., *Radiology.*
324. HOFFMAN, WILLIAM J., New York, N. Y., *Therapeutic Radiology.*
325. HOLLY, LELAND E., Muskegon, Mich., *Roentgenology.*
326. HOLMES, GEORGE W., Boston, Mass., *Radiology.*
327. HOLMES, RALPH W., Chillicothe, O., *Roentgenology.*
328. HOLSTON, JOEL D., Massillon, O., *Roentgenology.*
329. HOPKIRK, C. C., Santa Monica, Cal., *Roentgenology.*
330. HORRIGAN, ARTHUR J., Springfield, Mass., *Roentgenology.*
331. HOWARD, CAMPBELL, New York, N. Y., *Radiology.*
332. HOWARD, WILLIAM P., Albany, N. Y., *Roentgenology.*
333. HOWES, WILLIAM E., Brooklyn, N. Y., *Radiology.*
334. HUBENY, M. J., Chicago, Ill., *Roentgenology.*
335. HUFFORD, CLARENCE E., Toledo, O., *Radiology.*
336. HUNT, HOWARD B., Omaha, Neb., *Radiology.*
337. HUNT, RUSSELL R., Providence, R. I., *Roentgenology.*
338. HUNTER, JAMES W., JR., Norfolk, Va., *Radiology.*
339. HUTTON, FREDERICK C., Philadelphia, Pa., *Roentgenology.*
340. HYNES, WILLIAM P., Washington, D. C., *Therapeutic Radiology.*

341. IKEDA, KANO, St. Paul, Minn., *Roentgenology.*

342. Illick, H. Earl, New York, N. Y., *Roentgenology.*
343. Imboden, Harry M., New York, N. Y., *Roentgenology.*
344. Ingber, Irving S., San Francisco, Cal., *Radiology.*
345. Irwin, W. A., Detroit, Mich., *Radiology.*
346. Ivey, H. B., Goldsboro, N. C., *Radiology.*

347. Jaches, Leopold, New York, N. Y., *Radiology.*
348. Jackson, Byron H., Scranton, Pa., *Radiology.*
349. Jackson, John B., Kalamazoo, Mich., *Roentgenology.*
350. Jacobs, Lewis G., Jr., San Francisco, Cal., *Radiology.*
351. Jacox, Harold W., Pittsburgh, Pa., *Radiology.*
352. Jarre, Hans A., Detroit, Mich., *Radiology.*
353. Jenkinson, David L., Chicago, Ill., *Roentgenology.*
354. Jenkinson, E. L., Chicago, Ill., *Radiology.*
355. Johannesson, Carl J., Walla Walla, Wash., *Roentgenology.*
356. Johnson, Brantley M., Chicago, Ill., *Roentgenology*
357. Johnson, Clayton R., Los Angeles, Cal., *Roentgenology.*
358. Johnson, Ellsworth, Winchester, Va., *Roentgenology.*
359. Johnson, Jesse B., Galveston, Tex., *Radiology.*
360. Johnson, Sydney E., Louisville, Ky., *Diagnostic Roentgenology.*
361. Johnson, Vincent C., Ann Arbor, Mich., *Radiology.*
362. Johnston, Zoe Allison, Pittsburgh, Pa., *Therapeutic Radiology.*
363. Jones, Clifford F., Cleveland, O., *Roentgenology.*
364. Jones, Horace C., Detroit, Mich., *Radiology.*
365. Jones, William A., Kingston, Ont., *Radiology.*
366. Judson, Herbert A., Santa Monica, Cal., *Roentgenology.*
367. Judy, William S., Greenville, S. C., *Radiology.*

368. Kabakjian, Armen E., Lancaster, Pa., *Radiology.*
369. Kahn, Max, Baltimore, Md., *Roentgenology.*
370. Kalbfleisch, William K., Wheeling, W. Va., *Roentgenology.*
371. Kann, U. S., Binghamton, N. Y., *Radiology.*
372. Kaplan, Ira I., New York, N. Y., *Therapeutic Radiology.*
373. Kaplan, Morris, New York, N. Y., *Diagnostic Roentgenology.*
374. Kapo, Peter J., Mahanoy City, Pa., *Radiology.*
375. Kasabach, Haig H., New York, N. Y., *Radiology.*
376. Keichline, John M., Huntingdon, Pa., *Radiology.*
377. Keith, David Y., Louisville, Ky., *Radiology.*
378. Kelley, Jacob S., Providence, R. I., *Roentgenology.*
379. Kellogg, John O., San Diego, Cal., *Radiology.*
380. Kelly, James F., Omaha, Neb., *Radiology.*
381. Kenning, John C., Detroit, Mich., *Radiology.*
382. Kerby, James P., Salt Lake City, Ut., *Roentgenology.*
383. Kerr, H. Dabney, Iowa City, Ia., *Radiology.*
384. Kesmodel, Karl F., Birmingham, Ala., *Radiology.*
385. Kidder, Frank W., Los Angeles, Cal., *Diagnostic Roentgeology.*
386. King, J. Cash, Memphis, Tenn., *Radiology.*
387. Kinney, Lyell C., San Diego, Cal., *Radiology.*
388. Kirkland, A. Stanley, St. John, N. B., *Roentgenology.*
389. Kirklin, B. R., Rochester, Minn., *Radiology.*
390. Klapproth, Herman, Halstead, Kans., *Roentgenology.*
391. Klein, William, New Brunswick, N. J., *Radiology.*
392. Koiransky, Gennady H., Long Island City, N. Y., *Diagnostic Roentgenology.*
393. Kolvoord, Theodore, Battle Creek, Mich., *Diagnostic Roentgenology.*
394. Kornblum, Karl, Philadelphia, Pa., *Radiology.*
395. Kraft, Ernest, New York, N. Y., *Roentgenology.*
396. Kuegle, F. H., Janesville, Wis., *Roentgenology.*
397. Kugler, Joseph C., Jackson, Mich., *Roentgenology.*
398. Kuhlman, Fred Y., Springfield, Ill., *Roentgenology.*

399. Lafferty, R. H., Charlotte, N. C., *Radiology.*
400. Landau, George M., Chicago, Ill., *Roentgenology.*
401. Landes, W. L. S., York, Pa., *Roentgenology.*
402. Langan, Paul C., Akron, O., *Diagnostic Roentgenology.*
403. Lange, Sidney, Cincinnati, O., *Radiology.*
404. Langer, Heinz, Pittsburgh, Pa., *Therapeutic Radiology.*
405. Lanman, Everett L., Battle Creek, Mich., *Roentgenology.*
406. Lape, C. Pearley, Buffalo, N. Y., *Diagnostic Roentgenology.*
407. Larkey, Charles J., Bayonne, N. J., *Diagnostic Roentgenology.*
408. Lattman, Isidore, Washington, D. C., *Radiology.*
409. Law, F. M., New York, N. Y., *Diagnostic Roentgenology.*
410. Lawrence, W. S., Memphis, Tenn., *Radiology.*
411. Lawson, John D., Sacramento, Cal., *Radiology.*
412. Leddy, E. T., Rochester, Minn., *Radiology.*
413. Ledoux, Alfred C., Evanston, Ill., *Radiology.*
414. Leef, Edward, San Francisco, Cal., *Radiology.*
415. LeFevre, Walter I., Cleveland, O., *Roentgenology.*
416. Lehman, J. S., Philadelphia, Pa., *Radiology.*
417. Leibert, Harry F., Bethlehem, Pa., *Roentgenology.*
418. Lenz, Maurice, New York, N. Y., *Therapeutic Radiology.*
419. Leonard, Ralph D., Boston, Mass., *Roentgenology.*
420. Leucutia, T., Detroit, Mich., *Radiology.*
421. Levene, George, Boston, Mass., *Roentgenology.*
422. Levi, Irwin P., Anniston, Ala., *Roentgenology.*
423. Levin, Alfred G., Miami, Fla., *Radiology.*
424. Le Vine, David, El Dorado, Ark., *Diagnostic Roentgenology.*
425. Levine, Samuel, Philadelphia, Pa., *Radiology.*
426. Levitin, Joseph, San Francisco, Cal., *Radiology.*
427. Levyn, Lester, Buffalo, N.Y., *Diagnostic Roentgenology.*
428. Lewis, Raymond W., New York, N. Y., *Diagnostic Roentgenology.*
429. Lhevine, Morris B., Tulsa, Okla., *Roentgenology.*
430. Liebman, Charles, Montreal, Que., *Roentgenology.*
431. Liljedahl, Elmer N., Los Angeles, Cal., *Roentgenology.*
432. Lingeman, Leslie R., Rochester, N. Y., *Roentgenology.*
433. Lipschultz, Oscar, Minneapolis, Minn., *Roentgenology.*
434. Litschgi, Joseph J., Chicago, Ill., *Roentgenology.*
435. Littig, L. V., Madison, Wis., *Radiology.*
436. Lochry, Ralph L., Indianapolis, Ind., *Roentgenology.*
437. Lockwood, Ira H., Kansas City, Mo., *Radiology.*
428. Loud, Norman W., New Britain, Conn., *Diagnostic Roentgenology.*
439. Lucas, Charles D., Charlotte, N. C., *Therapeutic Radiology.*
440. Lucinian, Joseph H., Miami, Fla., *Roentgenology.*
441. Lutz, Jeremiah F., York, Pa., *Roentgenology.*

442. MacColl, D. R., Los Angeles, Cal., *Roentgenology.*
443. MacComb, W. S., New York, N. Y., *Therapeutic Radiology.*
444. McCarthy, Humphrey L., Boston, Mass., *Roentgenology.*
445. McCarthy, Justin E., Cincinnati, O., *Radiology.*
446. McCaw, William W., Washington, D. C., *Radiology.*
447. McClanahan, Charles W., West Los Angeles, Cal., *Radiology.*
448. McClelland, Donald C., Lafayette, Ind., *Radiology.*
449. McClure, C. C., Nashville, Tenn., *Radiology.*
450. McCormick, Arthur F., Du Bois, Pa., *Roentgenology.*
451. McCormick, H. G., Laurel, Miss., *Roentgenology.*
452. McCormick, William M., Du Bois, Pa., *Roentgenology.*
453. McCullough, John F., Pittsburgh, Pa., *Radiology.*
454. McDeed, Winfield G., Houston, Tex., *Roentgenology.*
455. McDermott, Joseph L., Kansas City, Mo., *Radiology.*
456. McElfatrick, George C., Wilmington, Del., *Roentgenology.*
457. McEuen, Harry B., Jacksonville, Fla., *Radiology.*
458. McGaughey, H. D., Joplin, Mo., *Radiology.*
459. McGee, Harry H., Savannah, Ga., *Roentgenology.*
460. McGehee, William H., Fresno, Cal., *Roentgenology.*
461. McGlothlan, Arthur B., St. Joseph, Mo., *Radiology.*
462. McGuffin, W. Herbert, Calgary, Alberta, *Radiology.*
463. McHenry, Rupert K., Houston, Tex., *Roentgenology.*
464. McIntosh, Harriet C., New York, N. Y., *Roentgenology.*
465. McKinney, Joseph T., Roanoke, Va., *Roentgenology.*
466. McNabb, Atholl M., Ottawa, Ont., *Roentgenology.*
467. McNamee, Edgar P., Cleveland, O., *Diagnostic Roentgenology.*
468. McNutt, John R., Duluth, Minn., *Radiology.*
469. McPeak, Clarence N., Fitchburg, Mass., *Roentgenology.*
470. McPeak, Edgar M., Washington, D. C., *Radiology.*

471. MACMILLAN, A. S., Boston, Mass., *Diagnostic Roentgenology.*
472. MAGRUDER, L. FREELAND, Norfolk, Va., *Radiology.*
473. MAHRER, HERBERT A., Cleveland, O., *Roentgenology.*
474. MAIER, ROE J., Chicago, Ill., *Radiology.*
475. MALCOLMSON, PATRICK H., Edmonton, Alberta, *Radiology.*
476. MALONE, LEANDER A., Terre Haute, Ind., *Radiology.*
477. MANDEVILLE, FREDERICK B., Richmond, Va., *Roentgenology.*
*478. MANGES, WILLIS F., Philadelphia, Pa., *Radiology.*
479. MARCHBANKS, S. S., Chattanooga, Tenn., *Radiology.*
480. MARKS, JOSEPH H., Fall River, Mass., *Roentgenology.*
481. MARQUIS, W. JAMES, Newark, N. J., *Roentgenology.*
482. MARTIN, CHARLES L., Dallas, Tex., *Radiology.*
483. MARTIN, JAMES M., Dallas, Tex., *Radiology.*
484. MARTIN, W. C., Louisville, Ky., *Roentgenology.*
485. MASON, CLAUDE H., El Paso, Tex., *Diagnostic Roentgenology.*
486. MASTERSON, JOHN J., Brooklyn, N. Y., *Roentgenology.*
487. MATLACK, JAMES A., Longmont, Col., *Diagnostic Roentgenology.*
488. MATTICK, WALTER L., Buffalo, N. Y., *Radiology.*
489. MAVER, WILLIAM W., Jersey City, N. J., *Roentgenology.*
490. MAWHINNEY, HARVEY N., Pittsburgh, Pa., *Diagnostic Roentgenology.*
491. MAY, ERNST A., East Orange, N. J., *Radiology.*
492. MAY, RAYMOND V., Cleveland, O., *Radiology.*
493. MAY, ROBERT J., Cleveland, O., *Radiology.*
494. MAYFIELD, CLAUD, Long Beach, Cal., *Diagnostic Roentology.*
495. MEADOWS, JAMES A., Birmingham, Ala., *Radiology.*
496. MEANS, HUGH J., Columbus, O., *Radiology.*
497. MEDELMAN, JOHN P., St. Paul, Minn., *Roentgenology.*
498. MELAND, ORVILLE N., Los Angeles, Cal., *Therapeutic Radiology.*
499. MELTZER, SAMUEL L., Portsmouth, O., *Diagnostic Roentgenology.*
500. MENDELSON, EMANUEL, Brooklyn, N. Y., *Roentgenology.*
501. MENEES, THOMAS O., Grand Rapids, Mich., *Radiology.*
502. MENVILLE, LEON J., New Orleans, La., *Radiology.*
503. MERCHANT, ALBERT K., Philadelphia, Pa., *Radiology.*
504. MERRILL, E. FORREST, New York, N. Y., *Roentgenology.*
505. MERRITT, E. A., Washington, D. C., *Radiology.*
506. MESIROW, SIDNEY D., Chicago, Ill., *Roentgenology.*
507. METER, EDWARD G., Reading, Pa., *Radiology.*
508. MEYER, KEITH T., Evansville, Ind., *Roentgenology.*
509. MEYER, WILLIAM H., New York, N. Y., *Radiology.*
510. MILES, JOHN M., New Orleans, La., *Diagnostic Roentgenology.*
511. MILKMAN, LOUIS A., Scranton, Pa., *Roentgenology.*
512. MILLER, HARRY A., Baltimore, Md., *Radiology.*
513. MILLWEE, ROBERT H., Dallas, Tex., *Radiology.*
514. MING, CHARLES M., Okmulgee, Okla., *Radiology.*
515. MINOR, EDWARD G., Detroit, Mich., *Diagnostic Roentgenology.*
516. MOFFAT, F. J., Medford, Ore., *Diagnostic Roentgenology.*
517. MOLONEY, ALBERT M., Boston, Mass., *Roentgenology.*
518. MOORE, ALEXANDER B., Washington, D. C., *Radiology.*
519. MOORE, CLAUDE, Washington, D. C., *Radiology.*
520. MOORE, DANIEL M., Monroe, La., *Roentgenology.*
521. MOORE, JOHN J., San Francisco, Cal., *Radiology.*
522. MOORE, PAUL D., Muncie, Ind., *Radiology.*
523. MOORE, SHERWOOD, St. Louis, Mo., *Radiology.*
524. MOORE, VERNOR M., Grand Rapids, Mich., *Radiology.*
525. MORRISON, MURRAY C., London, Ont., *Radiology.*
526. MORRISON, SIDNEY L., Boston, Mass., *Roentgenology.*
527. MORSE, RUSSELL W., Minneapolis, Minn., *Roentgenology.*
528. MORTON, S. A., Milwaukee, Wis., *Radiology.*
529. MOXNESS, BENNIE A., Northampton, Mass., *Diagnostic Roentgenology.*
530. MUELLER, W. K., St. Louis, Mo., *Diagnostic Roentgenology.*
531. MULLIGAN, PETER B., Ashland, Pa., *Roentgenology.*
532. MURPHY, G. W., Asheville, N. C., *Roentgenology.*
533. MURPHY, JOHN T., Toledo, O., *Radiology.*
534. MYERS, RALPH E., Oklahoma City, Okla., *Radiology.*
535. NASLUND, AMES W., St. Paul, Minn., *Roentgenology.*
536. NATHANSON, LOUIS, Brooklyn, N. Y., *Radiology.*
537. NELSON, PETER A., Chicago, Ill., *Therapeutic Radiology.*
538. NESSA, N. J., Sioux Falls, S. D., *Roentgenology.*
539. NEWCOMET, WILLIAM S., Philadelphia, Pa., *Radiology.*
540. NEWELL, ROBERT R., San Francisco, Cal., *Radiology.*
541. NICHOLS, B. H., Cleveland, O., *Radiology.*
542. NICHOLS, HAROLD E., Seattle, Wash., *Diagnostic Roentgenology.*
543. NORDIN, GUSTAF T., Minneapolis, Minn., *Radiology.*
544. O'BANNON, R. P., Fort Worth, Tex., *Radiology.*
545. O'BOYLE, CYRIL P., Philadelphia, Pa., *Diagnostic Roentgenology.*
546. O'BRIEN, FREDERICK W., Boston, Mass., *Radiology*
547. O'CONNELL, ANDREW E., Worcester, Mass., *Roentgenology.*
548. O'NEILL, JOHN R., San Francisco, Cal., *Roentgenology.*
549. OECHSLIK, WALDO R., Olive View, Cal., *Diagnostic Roentgenology.*
550. OGDEN, RALPH T., Hartford, Conn., *Radiology.*
551. OLIN, HARRY, Chicago, Ill., *Roentgenology.*
552. ORNDOFF, B. H., Chicago, Ill., *Radiology.*
553. ORR, CLIFFORD R., Buffalo, N. Y., *Radiology.*
554. OSMOND, JOHN D., Cleveland, O., *Roentgenology.*
555. OSMOND, LESLIE H., Pittsburgh, Pa., *Radiology.*
556. OSSIP, ABRAHAM, New York, N. Y., *Diagnostic Roentgenology.*
557. OSTRUM, HERMAN W., Philadelphia, Pa., *Roentgenology.*
558. OURIAN, ADOM K., New York, N. Y., *Diagnostic Roentgenology.*
559. OVERGAARD, ANDERS P., Omaha, Neb., *Radiology.*
560. OWEN, ARTHUR K., Topeka, Kans., *Roentgenology.*
561. OWEN, COLIN C., San Bernardino, Cal., *Roentgenology.*
562. PAINE, ROBERT, Memphis, Tenn., *Radiology.*
563. PALLEN, CONDE DES., Rochelle Park, N. J., *Therapeutic Radiology.*
564. PALMER, DORWIN L., Portland, Ore., *Radiology.*
565. PANCOAST, HENRY K., Philadelphia, Pa., *Radiology.*
566. PARKER, CARL H., Pasadena, Cal., *Roentgenology.*
567. PARMELEE, B. M., Bridgeport, Conn., *Radiology.*
568. PARRY, LEO D., Easton, Pa., *Radiology.*
569. PAUL, LESTER W., Madison, Wis., *Diagnostic Roentgenology.*
570. PAWLING, JESSE R., Watertown, N. Y., *Diagnostic Roentgenology.*
571. PEDEN, JOSEPH C., St. Louis, Mo., *Roentgenology.*
572. PEIRCE, CARLETON B., Ann Arbor, Mich., *Radiology.*
573. PENDERGRASS, E. P., Philadelphia, Pa., *Radiology.*
574. PENDERGRASS, ROBERT C., Americus, Ga., *Roentgenology.*
575. PERCIVAL, MILTON F., Philadelphia, Pa., *Radiology.*
576. PERKINS, CHARLES W., Norwalk, Conn., *Roentgenology.*
577. PERLBERG, HARRY J., Jersey City, N. J., *Diagnostic Roentgenology.*
578. PERLEY, ARTHUR E., Waterloo, Ia., *Radiology.*
579. PERRY, GENTZ, Evanston, Ill., *Radiology.*
580. PETERS, CHESTER M., Canton, O., *Radiology.*
581. PETERSON, CHARLES H., Roanoke, Va., *Roentgenology.*
582. PETERSON, GEORGE E., Waukesha, Wis., *Radiology.*
583. PETERSON, V. L., Charleston, W. Va., *Radiology.*
584. PETRIE, E. A., St. Johns, N. B., *Roentgenology.*
585. PETTIT, ROSWELL T., Ottawa, Ill., *Therapeutic Radiology.*
586. PFAHLER, G. E., Philadelphia, Pa., *Radiology.*
587. PHILIPS, HERMAN B., New York, N. Y., *Radiology.*
588. PHILLIPS, CLYDE C., Charlotte, N. C., *Radiology.*
589. PIERCE, HAROLD J., Terre Haute, Ind., *Radiology.*
590. PIERSON, JOHN W., Baltimore, Md., *Radiology.*
591. PINDELL, MERL L., Los Angeles, Cal., *Diagnostic Roentgenology.*
592. PINES, JOHN A., Orlando, Fla., *Therapeutic Roentgenology.*
593. PIRIE, A. H., Montreal, Que., *Radiology.*
594. PITTS, THOMAS A., Columbia, S. C., *Roentgenology.*
*595. PODLASKY, HARRY B., Milwaukee, Wis., *Roentgenology.*

596. Pohle, E. A., Madison, Wis., *Therapeutic Radiology.*
597. Pomeranz, Maurice M., New York, N. Y., *Radiology.*
598. Pomeranz, Raphael, Newark, N. J., *Roentgenology.*
599. Pomeroy, Lawrence A., Cleveland, O., *Radium Therapy.*
600. Popoff, Constantine, Haverhill, Mass., *Diagnostic Roentgenology.*
601. Popp, Walter C., Rochester, Minn., *Therapeutic Radiology.*
602. Porter, Horace W., Jackson, Mich., *Radiology.*
603. Portmann, U. V., Cleveland, O., *Radiology.*
604. Post, Joseph W., Philadelphia, Pa., *Diagnostic Roentgenology.*
605. Potter, Carlton F., Syracuse, N. Y., *Roentgenology.*
606. Potter, Hollis E., Chicago, Ill., *Roentgenology.*
607. Potter, Roy P., Marshfield, Wis., *Roentgenology.*
608. Pound, Robert E., New York, N. Y., *Roentgenology.*
609. Powell, E. V., Temple, Tex., *Radiology.*
610. Powers, Martin T., Utica, N. Y., *Diagnostic Roentgenology.*
611. Powers, Richard T., Springfield, Mass., *Radiology.*
612. Powers, Robert A., Palo Alto, Cal., *Radiology.*
613. Price, R. J., Dayton, O., *Radiology.*
614. Prouty, J. V., Terre Haute, Ind., *Radiology.*
615. Putts, B. Swayne, Erie, Pa., *Roentgenology.*

616. Quick, Douglas, New York, N. Y., *Therapeutic Radiology.*
617. Quigley, D. T., Omaha, Neb., *Radium Therapy.*
618. Quimby, A. Judson, New York, N. Y., *Roentgenology.*
619. Quiney, James J., Easton, Pa., *Radiology.*
620. Quinlan, Catherine M., Santa Rosa, Cal., *Roentgenology.*

621. Raap, Gerard, Miami, Fla., *Radiology.*
622. Radding, Moses B., Elizabeth, N. J., *Diagnostic Roentgenology.*
623. Rathbone, Ralph R., Washington, D. C., *Radiology.*
624. Rauschenbach, Charles W., Hammond, Ind., *Roentgenology.*
625. Ravold, Henry J., St. Joseph, Mo., *Radiology.*
626. Ray, William B. G., Pittsburgh, Pa., *Roentgenology.*
627. Reaves, Hugh G., Knoxville, Tenn., *Roentgenology*
628. Reed, Charles B., Newburgh, N. Y., *Roentgenology.*
629. Reeves, Robert J., Durham, N. C., *Radiology.*
630. Reiley, William E., Clearfield, Pa., *Radiology.*
631. Reineke, Harold G., Cincinnati, O., *Roentgenology.*
632. Reitter, George S., East Orange, N. J., *Radiology.*
633. Remer, John, New York, N. Y., *Therapeutic Roentgenology.*
634. Rendich, Richard A., Brooklyn, N. Y., *Roentgenology.*
635. Reynolds, Lawrence, Detroit, Mich., *Radiology.*
636. Rhinehart, Darmon A., Little Rock, Ark., *Roentgenology.*
637. Rhudy, Booker E., Greensboro, N. C., *Roentgenology.*
638. Rice, Frank M., San Francisco, Cal., *Roentgenology.*
639. Richards, Charles M., San Jose, Cal., *Radiology.*
640. Rigler, Leo G., Minneapolis, Minn., *Radiology.*
641. Ritvo, Max, Boston, Mass., *Radiology.*
642. Ritzman, Allen Z., Harrisburg, Pa., *Roentgenology.*
643. Roberts, Douglas J., Hartford, Conn., *Radiology.*
644. Robin, Nathaniel H., Hempstead, N. Y., *Roentgenology.*
645. Robin, Percival A., Hempstead, N. Y., *Roentgenology.*
646. Robins, Samuel A., Boston, Mass., *Roentgenology.*
647. Robinson, G. Allen, New York, N. Y., *Therapeutic Radiology.*
*648. Robinson, Ralph V., Pittsburgh, Pa., *Roentgenology.*
649. Robinson, Walter W., Memphis, Tenn., *Roentgenology.*
650. Rodenbaugh, Frederick H., San Francisco, Cal., *Radiology.*
651. Rodgers, Floyd D., Columbia, S. C., *Radiology.*
652. Rodick, J. C., New Orleans, La., *Roentgenology.*
653. Rodriquez, Juan, Fort Wayne, Ind., *Radiology.*
*654. Roland, Marion M., Oklahoma City, Okla., *Therapeutic Radiology.*
655. Rork, Lee W., Hastings, Neb., *Roentgenology.*
656. Rose, Cassie B., Chicago, Ill., *Radiology.*
657. Rosenbaum, George, Philadelphia, Pa., *Diagnostic Roentgenology.*
658. Rosh, Rieva, New York, N. Y., *Therapeutic Radiology.*
659. Rousseau, J. P., Winston Salem, N. C., *Radiology.*
660. Rowe, Edward W., Lincoln, Neb., *Radiology.*
661. Rowell, Edward E., Stamford, Conn., *Roentgenology.*
662. Rubenfeld, Sidney, New York, N. Y., *Therapeutic Radiology.*
663. Rubert, Samuel R., Chicago, Ill., *Roentgenology.*
664. Rude, Joe C., New York, N. Y., *Radiology.*
665. Rudisill, Hillyer, Jr., Charleston, S. C., *Radiology.*
666. Ruggles, Howard E., San Francisco, Cal., *Roentgenology.*
667. Rulison, Foster C., Syracuse, N. Y., *Roentgenology.*
668. Rutledge, Clifford P., Shreveport, La., *Radiology.*
669. Rypins, Edwin L., Bloomington, Ill., *Radiology.*

670. Samuel, Ernest C., New Orleans, La., *Roentgenology.*
671. Sanderson, Stevens S., Detroit, Mich., *Radiology.*
672. Sante, L. R., St. Louis, Mo., *Radiology.*
673. Sargent, William H., Oakland, Cal., *Radiology.*
674. Schenck, Samuel George, Brooklyn, N. Y., *Radiology.*
675. Schimmelpfennig, R. D., Montclair, N. J., *Roentgenology.*
676. Schmidt, Ernst A., Denver, Col., *Radiology.*
677. Schmitz, Henry, Chicago, Ill., *Therapeutic Radiology.*
678. Schnack, A. G., Honolulu, Hawaii, *Radiology.*
679. Schnoebelen, Paul C., St. Louis, Mo., Roentgenology.
680. Schons, Edward, St. Paul, Minn., *Radiology.*
681. Schumacher, F. L., Pittsburgh, Pa., *Radiology.*
682. Schwartz, Charles W., New York, N. Y., *Roentgenology.*
683. Schwartz, Irving, New York, N. Y., *Diagnostic Roentgenology.*
684. Scott, Clifton R., New Haven, Conn., *Roentgenology.*
685. Scott, Wilhelmina S., Lancaster, Pa., *Radiology.*
686. Seeds, Asa E., Spokane, Wash., *Radiology.*
687. Shapiro, Abraham V., Brooklyn, N. Y., *Diagnostic Roentgenology.*
688. Shapiro, William M., Toledo, O., *Radiology.*
689. Sharpe, A. Maxwell, Chester, Pa., *Roentgenology.*
690. Shaw, W. McL., Jacksonville, Fla., *Roentgenology.*
691. Shebesta, Emil M., Detroit, Mich., *Radiology.*
692. Sheldon, Francis B., Stockton, Cal., *Radiology.*
693. Sheridan, William M., Spartanburg, S. C., *Radiology.*
*694. Sherman, Herbert deG., White Plains, N. Y., *Diagnostic Roentgenology.*
695. Sherrick, Earl C., Connellsville, Pa., *Diagnostic Roentgenology.*
696. Shiflett, E. Lee, Louisville, Ky., *Roentgenology.*
697. Shore, O. J., Detroit, Mich., *Diagnostic Roentgenology.*
698. Shoulders, H. S., Nashville, Tenn., *Radiology.*
699. Shulman, Simon, Far Rockaway, N. Y., *Roentgenology.*
700. Sichler, Harper G., Lafayette, Ind., *Radiology.*
701. Sickels, Thomas N., Watertown, N. Y., *Roentgenology.*
702. Siefert, Alfred C., Oakland, Cal., *Radiology.*
703. Sigman, Frederick G., Salisbury, N. C., *Roentgenology.*
704. Sims, George P., Columbus, O., *Therapeutic Radiology.*
705. Skinner, Edward H., Kansas City, Mo., *Radiology.*
706. Skomp, C. E., Denver, Col., *Diagnostic Roentgenology.*
707. Smedal, M. I., Boston, Mass., *Radiology.*
708. Smith, Arthur B., La Jolla, Cal., *Diagnostic Roentgenology.*
709. Smith, B. B., Buffalo, N. Y., *Diagnostic Roentgenology.*
710. Smith, Jerome H., San Angelo, Tex., *Radiology.*
711. Smith, Lester A., Indianapolis, Ind., *Radiology.*
712. Smith, Richard L., Grand Rapids, Mich., *Radiology.*
713. Smith, R. Manges, Philadelphia, Pa., *Roentgenology.*
714. Smith, Roscoe L., Lincoln, Neb., *Radiology.*
715. Smyth, Thomas L., Allentown, Pa., *Roentgenology.*
716. Snead, Lawrence O., Richmond, Va., *Roentgenology.*
717. Snow, Henry, Dayton, O., *Radiology.*
718. Snow, William, New York, N. Y., *Roentgenology.*
719. Snure, Henry, Los Angeles, Cal., *Roentgenology.*
720. Soiland, Albert, Los Angeles, Cal., *Radiology.*
721. Solis-Cohen, Leon, Philadelphia, Pa., *Radiology.*
722. Sosman, M. C., Boston, Mass., *Radiology.*
723. Soule, Arthur B., Jr., Burlington, Vt., *Roentgenology.*
724. Spackman, Edgar W., Philadelphia, Pa., *Radiology.*
725. Spangler, Davis, Dallas, Tex., *Radiology.*

726. SPENCER, HUNTER B., Lynchburg, Va., *Roentgenology.*
727. SPENCER, JACK, Boston, Mass., *Radiology.*
728. SPILLMAN, RAMSAY, New York, N. Y., *Diagnostic Roentgenology.*
729. SPILMAN, HAROLD A., Ottumwa, Ia., *Roentgenology.*
730. SPINZIG, EDGAR W., St. Louis, Mo., *Radiology.*
731. SPROULL, JOHN, Haverhill, Mass., *Roentgenology.*
732. SQUIRE, FAY H., Chicago, Ill., *Roentgenology.*
733. STAFFORD, OWEN R., Los Angeles, Cal., *Therapeutic Roentgenology.*
734. STALL, ARTHUR H., Akron, O., *Radiology.*
735. STAMMEL, CHARLES A., Fort Benning, Ga., *Diagnostic Roentgenology.*
736. STARKS, DOROTHY J., Palo Alto, Cal., *Radiology.*
737. STARTZ, IRVING S., Elmhurst, N. Y., *Roentgenology.*
738. STAYTON, CHESTER A., Indianapolis, Ind., *Roentgenology.*
739. STECHER, WILLIAM R., Darby, Pa., *Radiology.*
740. STEEL, DAVID, Cleveland, O., *Roentgenology.*
741. STEINER, JOSEPH M., New York, N. Y., *Roentgenology.*
742. STEPHENSON, F. B., Denver, Col., *Radiology.*
743. STEVENS, R. H., Detroit, Mich., *Radiology.*
744. STEWART, HARRY M., Johnstown, Pa., *Radiology.*
745. STEWART, WENDELL, East St. Louis, Ill., *Diagnostic Roentgenology.*
746. STEWART, WILLIAM H., New York, N. Y., *Roentgenology.*
747. STONE, ROBERT S., San Francisco, Cal., *Roentgenology.*
748. STRAUSS, ABRAHAM, Cleveland, O., *Radium Therapy.*
749. STUART, LEON H., Tulsa, Okla., *Roentgenology.*
750. SUSSMAN, MARCY L., New York, N. Y., *Radiology.*
751. SUTHERLAND, CHARLES G., Rochester, Minn., *Diagnostic Roentgenology.*
752. SWENSON, PAUL C., New York, N. Y., *Roentgenology.*
753. SWOPE, OPIE W., Wichita, Kans., *Radiology.*
754. SYCAMORE, LESLIE K., Hanover, N. H., *Radiology.*

755. TABB, JOHN L., JR., Richmond, Va., *Roentgenology.*
756. TAFT, ROBERT B., Charleston, S. C., *Radiology.*
757. TALLEY, DANIEL D., JR., Richmond, Va., *Roentgenology.*
758. TAMARKIN, SAUL J., Youngstown, O., *Radiology.*
759. TAORMINA, LOUIS J., Brooklyn, N. Y., *Diagnostic Roentgenology.*
760. TAYLOR, CLIFFORD C., Indianapolis, Ind., *Radiology.*
761. TAYLOR, HENRY K., New York, N. Y., *Diagnostic Roentgenology.*
762. TAYLOR, RAYMOND G., Los Angeles, Cal., *Radiology.*
763. TAYLOR, RICHARD T., Los Angeles, Cal., *Radiology.*
764. TEITELBAUM, MEYER D., New Orleans, La., *Roentgenology.*
765. TEMPLETON, FREDERIC E., Chicago, Ill., *Radiology.*
766. THOMAS, M. A., Cleveland, O., *Radiology.*
767. THOMPSON, HAROLD B., Seattle, Wash., *Radiology.*
768. THURESSON, PAUL F., Riverside, Cal., *Roentgenology.*
769. TICE, GALEN M., Kansas City, Kans., *Radiology.*
770. TICHY, L. S., Chicago, Ill., *Roentgenology.*
771. TITTERINGTON, PAUL F., St. Louis, Mo., *Roentgenology.*
772. TREVES, NORMAN, New York, N. Y., *Therapeutic Radiology.*
773. TROJE, OSCAR R., Fairfield, Ala., *Radiology.*
774. TROSTLER, ISADOR S., Chicago, Ill., *Radiology.*
775. TROUP, RALPH L., Green Bay, Wis., *Radiology.*
776. TROXELL, WILLIAM C., Allentown, Pa., *Roentgenology.*
777. TUGGLE, ALLAN, New York, N. Y., *Radiology.*
778. TYLER, ALBERT F., Omaha, Neb., *Radiology.*

779. UDE, WALTER H., Minneapolis, Minn., *Roentgenology.*
780. ULBRICH, HENRY L., Detroit, Mich., *Roentgenology.*
781. ULLMANN, HENRY J., Santa Barbara, Cal., *Radiology.*
782. UNFUG, GEORGE A., Pueblo, Col., *Radiology.*
783. UNGER, ARTHUR S., New York, N.Y., *Roentgenology.*
784. UPSON, WILBUR O., Battle Creek, Mich., *Radiology.*

785. VAN ALLEN, HARVEY W., Springfield, Mass., *Radiology.*
786. VAN BUSKIRK, EDMUND M., Fort Wayne, Ind., *Roentgenology.*
787. VAN NUYS, ROSCOE G., Berkeley, Cal., *Radiology.*
788. VAN WINKLE, LEROY P., Brooklyn, N. Y., *Diagnostic Roentgenology.*
789. VASTINE, JACOB H., Philadelphia, Pa., *Radiology.*
790. VELKOFF, METODI, Fort Wayne, Ind., *Roentgenology.*
791. VIRDEN, C. EDGAR, Kansas City, Mo., *Radology.*
792. VOGT, EDGAR C., Boston, Mass., *Roentgenology.*
793. VOKE, EDWARD L., Akron, O., *Diagnostic Roentgenology.*
794. VOLDERAUER, JOHN C., Chicago, Ill., *Radiology.*
795. VOLTZ, ALBERT L., Richmond Hill, N. Y., *Roentgenology.*
796. VON POSWICK, GISELA, Scranton, Pa., *Roentgenology.*

797. WACHOWSKI, T. J., Chicago, Ill., *Roentgenology.*
798. WALKER, HOWARD M., Fort Worth, Tex., *Radiology.*
799. WALLACE, K. K., Norfolk, Va., *Radiology.*
800. WALTON, HENRY J., Baltimore, Md., *Radiology.*
801. WARDEN, MARINE R., Albuquerque, N. M., *Roentgenology.*
802. WARFIELD, CHESTER H., Wichita, Kans., *Roentgenology.*
803. WARREN, ALVA H., Malden, Mass., *Diagnostic Roentgenology.*
804. WASCH, MILTON G., Brooklyn, N. Y., *Radiology.*
805. WASSON, W. W., Denver, Col., *Radiology.*
806. WATERS, CHARLES A., Baltimore, Md., *Radiology.*
807. WATKINS, W. WARNER, Phoenix, Ariz., *Radiology.*
808. WEAVER, CLARENCE E., Detroit, Mich., *Roentgenology.*
*809. WEBB, JAMES A. H., Wichita, Kans., *Roentgenology.*
810. WEBER, HARRY M., Rochester, Minn., *Roentgenology.*
811. WEEKS, PAUL R., Denver, Col., *Radiology.*
812. WEISKOTTEN, W. OTTO, San Diego, Cal., *Roentgenology.*
813. WEITZNER, IMRE, New York, N. Y., *Diagnostic Roentgenology.*
814. WEITZNER, SAMUEL F., New York, N.Y., *Diagnostic Roentgenology.*
815. WENTWORTH, A. J., Mankato, Minn., *Radiology.*
816. WESCOTT, WILLIAM C., Atlantic City, N. J., *Radiology.*
817. WEST, JAMES H., Cleveland, O., *Radiology.*
818. WEST, THEODORE S., Port Chester, N. Y., *Roentgenology.*
819. WHEATLEY, FRANK E., Boston, Mass., *Radiology.*
820. WHEATLEY, LOUIS F., New Haven, Conn., *Roentgenology.*
821. WHELAN, CHARLES, Boston, Mass., *Radiology.*
822. WHITEHEAD, LAWTHER J., Richmond, Va., *Roentgenology.*
823. WHITLOCK, SIMON B., Norfolk, Va., *Roentgenology.*
824. WIDMANN, B. P., Philadelphia, Pa., *Radiology.*
825. WIGBY, PALMER E., Dallas, Tex., *Radiology.*
826. WILCOX, CLARK A., Wichita Falls, Tex., *Radiology.*
827. WILLIAMS, ARTHUR J., San Francisco, Cal., *Radiology.*
*828. WILLIAMS, FRANCIS H., Boston, Mass., *Radiology.*
829. WILLIAMS, LESTER J., Baton Rouge, La., *Radiology.*
830. WILLIAMS, P. A., Hempstead, N. Y., *Roentgenology.*
831. WILLY, RALPH G., Chicago, Ill., *Roentgenology.*
832. WINCHELL, A. VAUGHN, Rochester, N. Y., *Therapeutic Radiology.*
833. WILSON, HUGH M., New Haven, Conn., *Roentgenology.*
834. WILSON, JAMES E., Clarksburg, W. Va., *Diagnostic Roentgenology.*
835. WILSON, JOHN S., Monticello, Ark., *Therapeutic Radiology.*
836. WILSON, R. T., Austin, Tex., *Roentgenology.*
837. WILSON, STANLEY A., Lewiston, Me., *Roentgenology.*
838. WISSLER, J. E., Washington, D. C., *Radiology.*
839. WITHERS, SANFORD M., Denver, Col., *Therapeutic Radiology.*
840. WITWER, E. R., Detroit, Mich., *Radiology.*
841. WOOLFORD, JOSEPH S., Eureka, Cal., *Radiology.*
842. WRIGHT, CECIL S., Indianapolis, Ind., *Radiology.*
843. WURSTER, LLOYD E., Williamsport, Pa., *Roentgenology.*

844. YOUNG, BARTON R., Philadelphia, Pa., *Radiology.*
845. YOUNG, JAMES L. R., New York, N.Y., *Therapeutic Radiology.*

846. ZINK, OSCAR C., St. Louis, Mo., *Radiology.*
847. ZULICK, J. DONALD, Philadelphia, Pa., *Roentgenology.*

* Deceased

In addition to the above list there are a number who have passed part of the examination and will appear again for examination in other branches.

There will be two examinations conducted by the Board during 1937, the first in Atlantic City June 4, 5 and 6 and the second in Chicago September 10, 11 and 12. Those who contemplate appearing before the Board at either of these meetings should have their applications in the Secretary's office at least three months in advance of the meeting.

The Board is preparing a new roster of all diplomates. The Secretary would appreciate information concerning anyone who has changed his location since receiving his certificate.

B. R. Kirklin, M.D.,
Secretary-Treasurer,
The American Board of Radiology

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

D'EMIDIO, ANGELO SANTORO. Tumore cistico dell'emisfero cerebellare sinistro e della parte anteriore del verme. Recidiva operatoria. Radiumterapia efficace. (Cystic tumor of the left hemisphere of the cerebellum and the anterior part of the vermiform process. Operative recurrence. Effective radium therapy.) *Arch. di radiol.*, 1935, *11*, 231–238.

A case is described in a child aged seven. He complained of pain in the occipital region, vomiting, strabismus, staggering and ataxic gait; the latter increased until he could not walk or stand. Roentgen examination showed signs of tumor in the posterior fossa. He was given two series of roentgen treatments and after each improved but grew worse again. Operation was performed and showed a cystic tumor of the left hemisphere of the cerebellum which invaded the anterior part of the vermiform process. The surgeon removed the vermiform process and a part of the substance of the left cerebellar hemisphere with the electric bistoury. But it was necessary to leave a part of the tumor because of its close proximity to important vessels. The child seemed to improve for some months and was given another series of roentgen irradiations. But about eight months after the operation he began to have increasing difficulty in walking and examination of the eygrounds showed slight turgor of the vessels.

He was then given radium treatment. Four radium tubes each containing 10 mg. radium were placed in a Columbia paste apparatus modelled to the occipital region. The filter was 2 mm. platinum; the focus-skin distance 3 cm. The intention was to irradiate for twenty days but at the end of a week the child showed marked signs of intolerance and the treatment was discontinued. After two weeks' rest the child had recovered from the signs of intolerance and was again irradiated but with 11 tubes of 2 mg. radium each with a filter of 1 mm. platinum and a focus-skin distance of 2.5 cm. The third day he showed slight signs of intolerance and the treatment was again stopped. About six weeks later he was again irradiated at first with 11 and after three days with 15 tubes of 2 mg. each. He was therefore given a total of fifteen days' irradiation in forty-seven days. A month later he could stand on his feet. He was then given another irradiation of four days and two months later another irradiation of nine days. He continued to improve and was discharged in excellent condition with some neurological residues due to the operation and the prolonged intracranial hypertension. He is still in good condition almost two years after the last radium irradiation.

The author emphasizes the importance of irradiations of slight intensity, frequently repeated, in such cases and of close cooperation between surgeon and radiotherapist.—*Audrey G. Morgan.*

BRUNETTI, LODOVICO. Indicazioni e norme per l'assunzione di teleradiogrammi in craniologia. (Indications and projections for the taking of teleroentgenograms in craniology.) *Radiol. med.*, July, 1935, *22*, 673–689.

Many important details in craniometry can be studied only by means of teleroentgenography or hyperteleroentgenography. The advantage of teleroentgenography applied to anthropometric measurement of the skull is that it brings out the planes at different depths and projects them on a single plane, which is an extremely valuable contribution to anthropometric technique. The technique of the method is described, the different projections used defined and the practical application of the method to a number of anthropometrical problems reviewed. A number of roentgenograms and diagrammatic drawings illustrating the points discussed are given.—*Audrey G. Morgan.*

WOLKE, KNUT. Roentgen-anatomical studies of the labyrinth. *Acta radiol.*, 1935, *16*, 668–672.

So far as the author knows, the roentgen anatomy of the labyrinth has not been studied with contrast medium except by Stenvers in his projection. Lysholm and Runström have studied the labyrinth in three projections which are most useful but not with contrast medium. The author studied the labyrinth in Lysholm and

Runström's three projections and in a fourth practically identical with that of Stenvers. He sawed through several temporal bones and filled the semicircular canals, the vestibule and the basal convolution of the cochlea with red lead. The bones were then reconstructed and stereoscopic roentgenograms taken. In the lateral projection the skull lies on its side with a point corresponding to the middle of the external meatus in the center of the film. The central ray is centered with a cranial angle of 15°. In the axial orthoprojection in the lateral position the position is the same as above. A point about 2 cm. caudad from the external meatus is placed in the center of the film. The central ray is directed to this point with a cranial angle of 35°. In the axial projection the patient lies in such a way that a plane through the eye-ear line runs parallel with the plane of the film. The central ray bisects the line between the external auditory meati at a right angle with the eye-ear plane.

The pictures obtained in each projection are stereoscopic and by viewing the reproductions with divergent visual axes a good stereoscopic effect is obtained. Roentgenograms and diagrammatic drawings of the anatomy of the region are given.—*Audrey G. Morgan.*

Geschickter, Charles F. Tumors of the nasal and paranasal cavities. *Am. J. Cancer*, July, 1935, *24*, 637–660.

The majority of carcinomas of the nose arise in the region of the middle turbinate at the embryonic site of the outpouching of the sinuses, and are epidermal in type. In the nasopharynx, and more rarely in the nose and antrum, malignant epidermal cells from the mucous membrane are interspersed with lymphoid tissue, producing a variety of lymphodermal cancer usually referred to as lympho-epithelioma. The post-embryonic extensions of the epithelial membrane within the maxillary sinuses provide a source for epidermal carcinomas of the antrum. From the undifferentiated epithelium of the basal layers destined to form glandular appendages, cystic basal cell cancer and adenocracinoma arise. It is usually impossible at the time of their clinical recognition to determine whether malignant epithelial tumors of this region had their primary site within the nose or antrum.

The writer presents a classification and incidence of 211 tumors. Nineteen cases are benign epithelial tumors, 139 cases malignant epithelial tumors, 37 cases benign connective tissue tumors and 16 cases sarcomas. It is doubtful that the benign, inflammatory, nasal polyps undergo neoplastic or malignant change. More often the polypoid conditions are the result rather than the source of the malignancy. The writer goes into a detailed description of the many tumors found in the nasal and paranasal cavities. He divides the epidermal carcinomas into the squamous cell or transitional cell group, which are slowly growing tumors eroding bone and invading surrounding structures before they are clinically detected, and the lympho-epitheliomas which are most common in the nasopharynx.

The appendage cell tumors include cystic basal cell cancer, adenomas and cystadenomas, mucoid cysts and cholesteatomas, and adenocarcinoma. Of the benign connective tissue tumors, osteomas, angiomas, plasmocytomas and fibromas are the most common. In the malignant group of connective tissue tumors, spindle cell sarcoma and lymphosarcoma are at times found.

Numerous photographs and photomicrographs are included in this presentation which is most complete and cannot be fully comprehended unless read in detail.—*E. E. Downs.*

Neck and Chest

Picchio, Carlo. Appunti critici di anatomia radiografica e di semeiologia del collo. (Critical study of the roentgen anatomy and symptomatology of the neck.) *Radiol. med.*, Oct., 1935, *22*, 881–914.

The author has studied the normal anatomy of the bones of the larynx and trachea and the soft parts surrounding them on many roentgenograms of anatomical specimens and clinical cases. In regard to the bones he reviews the normal ossification of the cartilages and calls attention to the great variability in ossification in different individuals. He emphasizes particularly the islands of compact tissue that may appear in all the cartilages. Because of the great variability in the normal picture, the study of pathological changes in the skeleton of the larynx and pharynx by roentgen examination is not of the highest value.

But, on the other hand, the roentgen study of the surrounding soft parts is of the greatest value. The findings supplement the findings of clinical examination and often give even more detailed information. Sometimes it is the only

method of examination possible when conditions exist which render direct examination with the laryngeal mirror impossible. Exuberant growths are shown by changes in the transparency of the pharynx and larynx. Infiltrative processes of any kind, which are often not visible on direct examination because they do not involve the superficial layers of the mucosa, give typical roentgen pictures of swelling and deformity of the soft parts and of the outlines of the air passage. Ulcerations are readily visible, particularly when they affect the dorsum of the tongue, the epiglottis and the arytenoid eminences. Roentgenograms are given illustrating the normal findings and various pathological conditions including goiter and tumors of various kinds and at various sites.

The ventricles of Morgagni are by no means always visible.—*Audrey G. Morgan.*

MUCCHI, LUDOVICO. Una rara anomalia congenitala di posizione dell'aorta e dell'esofago. (An unusual congenital anomaly in the position of the aorta and the esophagus.) *Radiol. med.*, Dec., 1935, *22*, 1097–1104.

The author describes and illustrates with roentgenograms an unusual case of dextroposition of the arch of the aorta and anteposition of the esophagus. The patient was a man aged forty-six who had led a very active life and had never had any clinical symptoms attributable to this anomaly. The possible mechanism of the anomaly is discussed and also the interpretation of the different features of the roentgenogram.—*Audrey G. Morgan.*

BALLANTYNE, E. N. Coarctation of the aorta. *Am. J. Dis. Child.*, Sept., 1935, *50*, 642–648.

Ballantyne reports 3 cases of coarctation of the aorta which presented an interesting variety of detail both clinically and at necropsy. These were of the infantile type, the coarctation being situated between the origin of the left subclavian artery and the ductus arteriosus. Two were complete and the third was moderate. In the first two, the ductus was patent and was closed in the third. In the moderate case, there was a small opening in the interventricular septum. In one of the complete cases, there was a complete absence of the septum while in the other there were no cardiac anomalies.

Ballantyne states that a suggestion has been made that the better prognosis in the adult type is due to the position of the constriction forcing the establishment of a collateral circulation during early fetal life at a time when, incidentally, it can be most readily established. It has also been suggested that the classification of a given case as of the infantile or of the adult type should depend on the ability or inability of the constriction to produce obstruction of the flow of blood from the ductus arteriosus into the descending aorta in fetal life, owing to its position.

Comments are also made on the diagnosis and clinical and post-mortem incidence.—*R. S. Bromer.*

JOSEPHI, MARION G. Measurement of the size of the heart in normal children. *Am. J. Dis. Child.*, Oct., 1935, *50*, 929–944.

In this paper an effort is made to establish roentgenographic standards for the size of the normal heart in childhood by means of a statistical study of the relationship between various measurements of the heart and body. The following is the author's summary:

"A series of 418 observations on normal children is presented. These observations consist of values for height, weight, age, body surface area and the usual measurements of the heart. The measurements of the heart and those of the body have been correlated, and the highest degree of correlation has been shown to exist between the body surface area and the cardiac surface area. From the relationship of the body surface area to the cardiac surface area, a quotient has been derived which has been found to be a good measure of the series.

Correlation coefficients arranged in order of their degree of relationship are:

(a) Cardiac surface area to body surface area.
(b) Cardiac surface area to weight.
(c) Cardiac surface area to height.
(d) Cardiac surface area to age.
(e) Cardiac transverse diameter to weight.
(f) Cardiac transverse diameter to body surface area.
(g) Cardiac transverse diameter to chest diameter.
(h) Cardiac transverse diameter to height.

The formula of Hodges, Adams and Gordon for the prediction of the cardiac surface area has been applied to 100 children and the correlation between the observed cardiac surface

area and the predicted cardiac surface area has been found to be proper."—*R. S. Bromer.*

Semmola, Luigi. Rapporti morfologici tra cuore e stomaco studiati radiologicamente in cinquanta militari dal punto di vista della costituzione. (Morphological relations between the heart and stomach in 50 soldiers studied from the point of view of constitution.) *Arch. di radiol.*, 1935, *11*, 379–396.

The author reports an orthodiagraphic study of the heart and stomach made on 50 soldiers coming from different parts of Italy. He classifies them from the point of view of constitution according to the method of Viola. He finds that these two organs show morphological variations related to the type of constitution of the individual. The characteristics of the form and topography of the heart and stomach in these cases confirm the results reported in this field by other authors.

The constitutional morphological variations in the heart and stomach are correlated with each other, though there is not perfect correspondence in the degree of variation in the individual characteristics in the two organs.

There seems to be an exact correlation between the values of the angle of inclination of the two organs, which in the short and broad type are more obtuse than in the long and slender type. Very acute angles of inclination of the heart in the long and slender type are accompanied by correspondingly acute angles of inclination of the stomach and very obtuse angles of inclination of one of the organs in the short and broad type are accompanied by correspondingly obtuse angles in the other.

Tables showing the details of the results are given.—*Audrey G. Morgan.*

Browder, Jefferson, and DeVeer, J. Arnold. The varied pathologic basis for the symptomatology produced by tumors in the region of the pulmonary apex and upper mediastinum. *Am. J. Cancer*, July, 1935, *24*, 507–521.

The purpose of this paper is to point out, by means of 5 illustrative cases, that the symptomatology of malignant tumors located in the region of the pulmonary apex or upper mediastinum furnishes evidence only of the implication of various anatomical structures and is not indicative of the presence of a neoplasm of a particular pathological nature. The authors believe that the grouping of various captions of such symptoms as though they constituted a clinical or pathological entity is undesirable.

In 4 of the 5 cases described the Horner syndrome was present. Two of the cases were diagnosed histologically as metastatic carcinoma, 2 as primary epidermoid carcinoma and 1 as thymic carcinoma.

From the analysis of these cases and others cited from the literature, the authors conclude that it appears that the symptoms depend upon the implication chiefly of the following structures: portions of the brachial plexus or certain of its component spinal nerves, the cervical sympathetic trunk and the great vessels in the involved area. To these symptoms are added roentgenologic and physical signs consistent with the presence of tumor.

They believe that the symptom-complex cannot be considered to constitute either a clinical or pathological entity but serves only to indicate the presence of some malignant tumor in a rather restricted anatomical area.—*E. E. Downs.*

Bagnaresi, Giacomo. Influenza del decubito sull'aspetto radiografico e sugli spostamenti del lobo medio nell'infiltrazione pneumonica. (Effect of decubitus on the roentgen appearance and displacement of the middle lobe in pneumonic infiltration.) *Radiol. med.*, Oct., 1935, *22*, 915–928.

The author describes 2 cases of pneumonia of the middle lobe and discusses the differences made in the roentgenogram by changes in the position of the patient. Pneumonia causes changes in the size of the lobe and increases its specific gravity so that it changes its position with changes in the position of the patient. It makes pendulum movements around the hilus as a fulcrum. In the semi-sitting or supine position the lobe glides downward and backward. With the patient standing the lobe tends to glide backward and downward along the plane of the diaphragm so that it assumes a more vertical position. In the prone position the lobe tends to glide forward and upward. In the standing position the normal ray strikes the apex of the lobe and the outline is apt to be indistinct while with the patient in the prone position the normal ray strikes a thicker part of the lobe and the outline is more distinct and denser. A pleuritic exudate may exaggerate this roentgen appearance. These different ap-

pearances may be eliminated by varying the incidence of the normal ray.

In studying cases of pneumonia of the middle lobe and interlobar processes the patient should be examined in different positions and with different incidences of the normal ray.—*Audrey G. Morgan.*

DALL'ACQUA, VIRGILIO, and BELLI, MARIO. Ombre triangolari paramediastiniche basilari. (Triangular paramediastinal shadows at the base.) *Radiol. med.*, Nov., 1935, *22*, 977–993.

The authors discuss the significance of a triangular paramediastinal shadow at the base in the roentgenogram. From their experience, verified by a study of the literature, they state that this shadow may be caused by two things: either a pathological process in an accessory lower lobe or a blocked pleurisy in the postero-inferior part of the mediastinum. The former is by far the more frequent of the two.

Among the chief changes that occur in an accessory lower lobe and cause this shadow is bronchiectasis complicated by chronic bronchopneumonia. If there are ampullar dilatations of the bronchi without the parenchyma being affected, there may be a triangular shadow with a reticulate appearance. The authors report 3 cases of this unusual type, the pathological lobe being bilateral in one case and in the other two on the right side.

They then discuss the roentgen differential signs between an infiltration of an accessory lower lobe and a blocked postero-inferior mediastinal pleurisy. The most important signs are the following: (1) the characteristics of the pathological shadow; (2) the mobility of the shadow on respiratory movements; (3) the convexity or concavity of the hypotenuse of the triangle, and (4) the characteristics of the shadow in the lateral projections. They then discuss the clinical symptomatology and other methods of diagnosis, among which bronchography is the most important one.

There are other causes which may change the form of the cardiodiaphragmatic angle, such as retrocardiac aneurysms, enlargement of the left auricle, pericarditis, cold abscess from caries of the vertebrae, idiopathic dilatation of the esophagus and hernia of the esophageal hiatus in the diaphragm. None of these, however, give the characteristic triangular shadow that is caused by infiltration of an accessory lower lobe or by mediastinal pleurisy.—*Audrey G. Morgan.*

BENASSI, ENRICO. Reperto radiologico positivo da polmonite crupale prima del brivido. (Positive roentgen findings in croupous pneumonia before the chill.) *Radiol. med.*, Oct., 1935, *22*, 942–947.

There are very few roentgen descriptions in the literature at present of the initial stage of pneumonia and the author knows of only 2 cases of positive roentgen identification immediately after the chill. He describes a case in which he had occasion to make a roentgen examination in a heart patient and was able to demonstrate the opacification of the lung brought about by a beginning acute croupous pneumonia even before the chill had occurred. He was able to compare this roentgenogram with those he had taken before and with those that followed the resolution of the acute condition.

This early opacity, which was not seen in either the earlier or the later roentgenograms, consisted of quite an intense shadow immediately above the middle incisure, the lower edge of which was straight and very distinct, while the upper edge was convex and indistinct. Two hours after this examination the chill came on, followed by a typical acute croupous pneumonia of the right upper lobe. This case helps to support the theory of the bronchial origin of pneumonia rather than its hematogenous origin.

While in ordinary cases there would be no reason for making a roentgen examination before the chill, roentgen examinations in the early stages of the disease would probably prove of practical interest.—*Audrey G. Morgan.*

TONIOLO, GIUSEPPE. Sulle immagini pseudocavernose. (Pseudo-cavity images.) *Radiol. med.*, Nov., 1935, *22*, 1013–1022.

The author describes a case in a girl five years of age and illustrates it with roentgenograms. She was sent to the hospital with a diagnosis of pleurisy. Roentgen examination showed a bronchopneumonia at the right base with pleural adhesions. From the third to the eighth intercostal space there was a large round structure that looked like a cavity. Examination for tubercle bacilli was negative and the tuberculin reaction was negative. Twenty days from the

first examination this pseudo-cavity had disappeared completely. This is much too short a time for a real tuberculous cavity to heal. It was an encysted interlobar pneumothorax in which there were such complete adhesions of the interlobar fissure that it formed a closed cavity, into which air entered and gave the typical picture of a cavity.

If a picture which is apparently that of cavity disappears in from a minimum of twenty days to a maximum of fifty to sixty days it is safe to conclude that it is not a true tuberculous cavity. The author thinks that encysted interlobar pneumothorax is much more frequent than is generally believed; as the course of such a pneumothorax is benign it is very often not examined roentgenologically. In case such a pneumothorax is mistaken for cavity a series of roentgen examinations will readily clear up the diagnosis.—*Audrey G. Morgan.*

Jacobaeus, H. C. Case of severe bullous emphysema. *Acta radiol.*, 1935, *16*, 661–667.

A man aged forty-eight suffered a severe blow on the left side of the chest. A few months later he began to have shortness of breath on climbing stairs and on exertion. The author does not believe, however, that the trauma caused the bullous emphysema which was later discovered. He has not found trauma mentioned as the cause of this condition and from the fact that roentgenograms taken at this time were about the same as those taken four years later he believes the disease was of older date. This case was for a long time considered one of spontaneous pneumothorax. There was the typical picture of pneumothorax with weakened or absent breath sounds and increased resonance on percussion. The roentgenogram indicated pneumothorax with many fine adhesions in the upper and lower part of the pleural cavity. The author performed a thoracoscopy in order to determine the possibility of treatment by cauterizing the adhesions. He was surprised to find an enormous emphysematous cavity with many fine fibers passing from the lung to the chest wall, much more delicate than the finest adhesions seen in pneumothorax. These were without doubt remnants of the walls of the alveoli. There were no signs of infection in spite of the extensive damage to the parenchyma.

Though the diagnosis was cleared up in this case by the thoracoscopy the author would not recommend this procedure, particularly in cases of smaller emphysematous cavities. With such a large cavity as this the conditions are more nearly like those of ordinary pneumothorax.—*Audrey G. Morgan.*

Oliveri, Antonio. Morfologia costituzionale degli ili polmonari. (Constitutional morphology of the hila of the lungs.) *Radiol. med.*, Oct., 1935, *22*, 933–942.

The author studied the constitutional morphology of the hila of the lungs on about 500 roentgenograms of normal subjects of both sexes. He takes into account the relations that exist between the pulmonary vessels and the constitution of the lungs and heart in determining the form of the hilar shadow. He divides the subjects into the three fundamental types of constitution: the normolinear, the longilinear and the brevilinear. He finds that in normal subjects there are two chief types of the hilar-perihilar shadow, the longilinear and the brevilinear. The first is seen in subjects whose heart and lungs are of the longilinear type, the second in those in whom the heart and lungs are of the brevilinear type. In subjects in whom the type of constitution of the heart and lungs do not correspond, the type of hilar shadow is generally intermediate between the two mentioned above. A type that is absolutely normal for all normal individuals can hardly be described. Roentgenograms and diagrammatic outlines of the different types are given.—*Audrey G. Morgan.*

Wolff, Aage O. Contribution à la topographie des ganglions intrathoraciques surtout a l'égard de la tuberculose des ganglions bronchiques. (Topography of the intrathoracic glands, especially with reference to tuberculosis of the bronchial glands.) *Acta radiol.*, 1935, *16*, 675–681.

The author describes the anatomy of the intrathoracic glands, giving a diagram showing the localization of the different glands. He points out that the ordinary roentgenogram in the dorsoventral projection does not show these glands clearly. A roentgenogram in the right posterior oblique position, on the other hand, separates the hilar shadows and gives a clear image of the region. Roentgenograms in cases with calcified glands are reproduced which show that in this projection the hilar shadows are separated throughout their extent and no

longer masked by the thoracic organs. They show the relation of any process of infiltration to the spinal column and give a general view of the arrangement of the different gland groups. However, in fresh tuberculosis in children the fresh process is relatively circumscribed and does not give any direct shadow on the film either in the frontal projection or in the oblique projection described above.—*Audrey G. Morgan.*

LAURELL, HUGO. Der Nachweis minimaler, bei gewöhnlicher Lungenuntersuchung oft unsichtbarer Pleuraexsudate. Zugleich ein Beitrag zur Differentialdiagnose bei "lamellärer Pleuritis" und bei basaler Verschleierung. (Roentgen demonstration of exudates frequently invisible when ordinary methods of examination are used; study of differential diagnosis of lamellar pleurisy and basal cloudiness.) *Acta radiol.*, 1935, *16*, 691–704.

In order to demonstrate a small pleural exudate that cannot be seen on ordinary methods of examination the lung should be examined with the patient lying on the diseased side. As the author and Hjelm have pointed out before, a distinct mantle exudate will in that case become visible.

The author describes the method of differentiating a true lamellar pleurisy from physiological mantle shadows caused by the subpleural layers of muscle and fat. Among these sources of error the muscular subpleural shadow of Knutsson is decidedly the most important.

In some cases of bronchitis or beginning bronchopneumonia with atelectasis, the roentgen symptoms such as veiling of the bases and decreased mobility of the diaphragm on the diseased side may disappear completely after a deep respiration, particularly if the patient is lying on the normal side. In such cases the patient should be roentgenographed both seated and lying on both the normal and diseased sides. If these roentgenograms are interpreted in connection with the clinical findings it is much easier to interpret the obscurity at the base and determine whether the case is one of pleurisy, atelectasis or pneumonia, or perhaps a combination of these diseases.—*Audrey G. Morgan.*

GLIOZZI, S. M. Rilievi sul profilo ed in particolare sulle cosidette "pinzature" del diaframma. (Irregularities in the outline of the diaphragm.) *Arch. di radiol.*, 1935, *11*, 117–126.

It was formerly believed that the diaphragm under normal conditions showed a single curved line and that any other form was a manifestation of pathological changes in the muscles or the serous covering of the upper surface of the diaphragm. But many cases have been seen in which the diaphragm showed a mammillated outline, which has been variously described as a zig-zag outline, a tent-shaped outline, a delta image, etc. There may be two or a number of these protrusions. The author discusses their cause in connection with a number of cases which he describes in which they were seen after pneumothorax. They were formerly attributed to increased negative intrapleural pressure, but the author believes that changes in intrapleural pressure play only a secondary part in their causation. They may be caused by adhesions between the basal and diaphragmatic pleura, by digitations of the diaphgram or by various other factors, some of which the author admits are unknown.—*Audrey G. Morgan.*

KIRSHBAUM, JACK D. Myosarcoma of the diaphragm. *Am. J. Cancer*, Dec., 1935, *25*, 730–737.

Previous to this paper 6 cases have been reported of primary tumor involving the diaphragm, 3 being malignant neoplasms and 2 of these containing myogenic elements. The 2 cases included by the author were sarcoma of the diaphragm with extensive metastases. One tumor could be identified as leiomyosarcoma, while in the other tumor the character of the cells suggested a relation to striated muscle fibers.

The two types of malignant tumor derived from skeletal muscle fibers are discussed: the myoblastic sarcoma that originates from undeveloped muscle fibers, the myoblasts; the rhabdomyosarcoma, which is related to more mature striated muscle fibers. In contrast to the two malignant types there are the benign forms, the myoblast myoma and the rhabdomyoma.

There are probably many undifferentiated myogenic tumors originating from striated muscle diagnosed simply as sarcoma. Recognition of the earliest stages of differentiation of tumor cells toward striated muscle fibers will aid in identifying these rapidly growing tumors. The fact that in the myogenic tumor cell the tendency to differentiation remains rudimen-

tary probably accounts for the failure of cross striations to develop. The source of very undifferentiated rhabdomyosarcomas may be sought in isolated muscle fibers that have failed to differentiate fully during fetal life. Benign myoblastomas which are derived from undeveloped skeletal muscle do not tend to undergo sarcomatous transformation. They are usually of long duration and are composed of large cells with a light protoplasm containing numerous fine granules and a relatively small central nucleus. It may be necessary to stain these tumor sections with fat stains to differentiate them from xanthoma cells.—*E. E. Downs.*

ABDOMEN

Cola, Giuseppe. Ricerche radiologiche sui reflessi intersegmentari dell'apparato digerente. (Roentgen study of intersegmental reflexes in the digestive tract.) *Arch. di radiol.*, 1935, *11*, 217–230.

The author studied the movements of the appendix by means of roentgen examination. Roentgenograms of the digestive tract are reproduced. He found that there are a series of intersegmental reflexes acting between the different segments of the digestive tract and that the movements of filling and emptying of the stomach affect not only the colon and small intestine but also the appendix. The filling of the stomach immediately awakens the motor activity of the appendix which generally results in its partial or complete emptying. This reflex is present even under normal conditions but is particularly frequent and active in long appendices, in colitis and in some cases of chronic appendicitis.

It is hard to determine exactly the paths which these reflexes follow but it is important to know of these functional correlations which exist between the stomach and the various parts of the intestinal tract.—*Audrey G. Morgan.*

Militzer, Raymond E. Carcinoma of the stomach in identical twins. *Am. J. Cancer*, Nov., 1935, *25*, 544–550.

As far as can be determined, the author was unable to find any record of carcinoma of the stomach occurring simultaneously in uniovular twins. The brothers in this report had a marked resemblance and both, at the age of seventy, became conscious of a gastric derangement. The symptoms occurred about a month apart and both were admitted to the hospital within four months after their first complaint. The microscopic diagnosis was adenocarcinoma of the stomach. In both the disease progressed rapidly. Of incidental interest was the history of bilateral inguinal hernia in each with recurrence in both after herniorrhaphy. The occurrence of similar simultaneous and symmetrical carcinomata in identical twins tends to give further confirmation to the theory of the genetic origin of these tumors.—*E. E. Downs.*

Brulé, Marcel; Cain, Andre; Moulonguet, Pierre; Hillemand, Pierre; and Aubrun, W. Les tumeurs villeuses de l'estomac. (Villous tumors of the stomach.) *Presse méd.*, March 21, 1936, *44*, 482–485.

The authors had the good fortune to observe, within a few weeks, 2 gastric tumors of unusual appearance. The roentgen examination in the first revealed a polycyclic defect the size of a large orange which was thought to be due to a benign lesion. In the second likewise there was a polycyclic appearance, the tumor being of about the same size, but there was a definite induration with fixation of the adjacent parts of the stomach, from which it was concluded that the tumor must represent a malignant degeneration. The histologic examination in both instances revealed, to the authors' surprise, a type of neoplasm which approaches the villous tumors as observed in the rectum. After describing both cases in detail by using roentgenograms, specimens and numerous histologic sections for illustration and after discussing at length the villous nature of these lesions, the authors arrive briefly at the following conclusions: It is legitimate to describe at the level of the stomach villous tumors which present characteristics similar to those observed in the rectum, the chief among them being the fact that after a certain phase of benignity a malignant degeneration may follow. The separation of such a group has not only a histologic and pathologic significance but is important also from the clinical, therapeutic and prognostic standpoint. By analogy with the villous tumors of the rectum which are known for their lack of recurrence after local removal, even when showing malignant degeneration, the authors raise the question whether some of the gastric cancers which heal without recurrence following gastrectomy do not in fact represent degener-

ated villous tumors in which the neoplastic invasion remained localized to the superficial layers of the stomach.—*T. Leucutia.*

BAGNARESI, G., and BARGI, L. Radiologica e clinica dei diverticoli duodenali. (Clinical and roentgen study of diverticula of the duodenum.) *Arch. di radiol.*, 1935, *11*, 280–324.

Sixteen cases of diverticulum of the duodenum are described and illustrated with roentgenograms. Two cases of false diverticula due to abnormal kinking of the duodenum are also described. The majority of these cases were ones of acquired diverticulum. The differences in the roentgen pictures of the diverticula are mostly due to different stages of filling of the cavities. The filling and emptying of the diverticula is dependent on the peristalsis of the duodenum rather than on contractions of the walls of the diverticulum itself. The lacunar images seen in some of the diverticula seem to have been caused by a penetration of the mucous membrane of the duodenum into the cavity rather than by a folding of the mucous membrane of the diverticulum itself.

The symptomatology of duodenal diverticulum is not very characteristic. In fact, many of them do not cause any symptoms at all and it is only by roentgen examination that the number of cases diagnosed during life can be made to approximate the number found on postmortem examination. When they do cause symptoms it is generally because of inflammation or traction from adhesions. The symptomatology when it exists may be confused with that of ulcer, cholecystitis or malignant tumor and it requires observation over a long period of time to make a clinical diagnosis of diverticulum.

Surgical treatment is generally not justified unless there is inflammation or an association with some other abdominal disease. If operation is performed a careful examination should be made in order to find and treat any other associated disease. Such associated conditions, for example gastroduodenal ulcer, pancreatitis, perivisceritis, gastroduodenitis, cholecystitis, etc., seem to favor the development of diverticula. Medical treatment directed toward keeping the diverticulum empty helps to prevent complicating inflammation. The administration of a suspension of barium before meals seems to be effective in coating the wall of the diverticulum and preventing the accumulation of food and consequent inflammation.—*Audrey G. Morgan.*

DA EMPOLI, GIOVANNI. Le dolicocolie totali del colon sinistra. (Total dolichocolia of the left colon.) *Arch. di radiol.*, 1935, *11*, 239–248.

Four cases of elongation of the whole of the left colon are described and illustrated with roentgenograms. The patients were 21, 45, 52 and 76 years of age. The first was without doubt a case of congenital dolichocolon, for, in addition to the abnormal length of the colon, it showed an abnormal course from defective rotation in embryonic life. The other cases did not show definite signs of a congenital origin and were probably acquired. In these patients the chief symptom is constipation, dating sometimes from childhood, sometimes beginning at a later period. The most frequent symptoms of distal dolichocolon are constipation, meteorism, pain localized over the affected part of the intestine, and fever, usually of the hectic type. Signs that are less constant are melena, transitory diarrhea, masses in the abdomen due to stagnation of feces. In 1,000 roentgen examinations of the digestive tract the author found dolichocolon in 3 per cent.—*Audrey G. Morgan.*

SKELETAL SYSTEM

KOTTMEIER, HANS-LUDVIG. Knochenveränderungen bei malignen Uterus- und Ovarialtumoren. (Bone changes in malignant tumors of the uterus and ovaries.) *Acta radiol.*, 1935, *16*, 275–294.

After a short review of the literature the author describes the cases of malignant tumor of the uterus and ovaries which have been treated by irradiation at the Serafim Hospital in Stockholm and then examined roentgenologically because there was a suspicion of changes in the bones. There are three kinds of bone changes in malignant tumors of the uterus and ovaries: (1) destruction by pressure from tumors; (2) destruction by gland metastases, and (3) true bone metastases. He finds that metastases in the bones are not as infrequent in cases treated by radiotherapy as is indicated in earlier foreign statistics mostly based on cases not treated by irradiation. Adenocarcinoma of the cervix has a greater tendency to produce metastases than pavement epithelial cancer has. Bone metastases in cancer of the uterus are decidedly osteolytic, while in cancer

of the ovary they may be both osteolytic and osteoplastic. In order to differentiate between osteoporosis and metastases, structural pictures must be obtained. A description is given of the technique used at the Serafim hospital for photography of the spinal column and pelvis. Good structural pictures of the vertebrae can be obtained even in cases of marked osteoporosis by using a greater focal distance, a longer exposure period, lower tension and a Lysholm grid. Areas of destruction caused by lymph gland metastases are to be looked for in the region of the sacroiliac joint and the greater sciatic notch and special roentgenograms are often necessary for the examination of this region.

In order to differentiate between bone metastases and other pathological conditions in the bones close cooperation between the clinician and the roentgenologist is necessary.—*Audrey G. Morgan.*

Lupacciolu, Giovanni. Fratture del rachide cervicale all'indagine radiologica. (Roentgen examination in fracture of the cervical spinal column.) *Radiol. med.*, June, 1935, *22*, 529–562.

The author briefly discusses the technique of roentgen examination of the cervical spinal column and then reviews the most interesting cases of fracture of this segment of the spinal column that he has seen in his practice at the Littorio and Umberto I hospitals in Rome. Ten cases are described in detail and illustrated with roentgenograms. He first discusses traumatic lesions of the atlas and axis, especially their pathogenesis and roentgen diagnosis, quoting extensively from the literature on the subject. He then discusses traumatic lesions of the rest of the cervical column. He reviews the anatomical structure of the intervertebral disc and its physiopathology showing that lesions of the disc are a factor of the first importance in fractures of this part of the spinal column.—*Audrey G. Morgan.*

Wachner, G. Über die Lymphogranulomatose des Sternums. (Lymphogranulomatosis of the sternum.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, May, 1935, *51*, 492.

Wachner reports 3 cases of secondary involvement of the sternum by lymphoblastoma as one of the more unusual sites of osseous involvement in this disease. In differential diagnosis the response of the sternal lesion to radiotherapy is of greatest value.—*E. T. Leddy.*

Stenström, Bo. Über Phalangetuberkulose bei älteren Individuen. (Tuberculosis of the phalanges in older individuals.) *Acta radiol.*, 1935, *16*, 471–477.

Tuberculosis of the fingers generally occurs in the form of spina ventosa in children. Some cases have been described in adults and elderly individuals, however. The author describes 3 such cases, in a man of twenty-nine, a man of seventy-six and a woman of sixty-nine. In the first case the patient had had pain in the middle phalanx of the right index finger for about six weeks. The finger was swollen. Roentgen examination showed destructive changes in the basal half of the affected phalanx. There was spontaneous fracture and collapse of the bone and periosteal irritation around it. A diagnosis could not be made from the roentgen picture alone but as the clinical symptoms suggested sarcoma the finger was exarticulated. Histological examination showed the bone of the affected phalanx destroyed and replaced by granulation tissue in which there were accumulations of epithelioid cells and giant cells of the Langhans type. The histological picture was that of tuberculosis but as no bacilli could be demonstrated it is possible that it may have been syphilis. No tumor tissue was found.

In the second case the patient had had pain in the left middle finger for three weeks; the finger was swollen. The roentgenogram showed periostitis with deposits all around the phalanx. There was no focal destruction but the bone was thinned and in the affected parts no definite cortex could be found. The pictures suggested inflammation rather than tumor. The finger was removed. After decalcification the bone showed pronounced tuberculous osteitis. The third case was very similar to the second both in roentgen and histological findings.

There is little material in the literature on such cases. It is impossible by roentgen examination alone to make a differential diagnosis between ordinary chronic osteomyelitis and tuberculous osteitis. The history and clinical findings must be taken into consideration in making a differentiation from osteomyelitis, syphilis, tumor and benign lymphogranuloma. —*Audrey G. Morgan.*

HERLYN, K.-E. Über Fehldiagnosen bei Spondylitis unter Berücksichtigung atypischen Sitzes, der Blockwirbildung und der Spondylitis luica. (Errors in diagnosis of spondylitis with special reference to atypical localization, ankylosis, and spondylitis luica.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, May, 1935, *51*, 521.

In a follow-up examination of 35 patients out of a group of 115 who had been treated conservatively for tuberculous spondylitis, 13 in a period of six to eight years were found to have been mis-diagnosed. These 13 cases showed that many nonspecific diseases may have the false appearance of spondylitis. The author discusses in detail the differential diagnosis of these lesions.—*E. T. Leddy.*

HARRIS, R. I. The early diagnosis of Pott's disease. *Am. Rev. Tuberc.*, 1934, *29*, 223–229.

Pott's disease, being hematogenous in origin, appears only in patients who have tuberculosis elsewhere. Though not a common disease, it is a potential possibility in every patient under treatment for tuberculosis.

The symptom most valuable for early diagnosis is spinal pain, increased by activity and weight bearing, and relieved by rest. The remaining symptoms and signs upon which a diagnosis may be made are muscle spasm, deformity, abscess formation, characteristic roentgenographic appearance and paraplegia.

The disease exists for a varying period of time before deformity appears, dependent partly upon the segment involved, partly upon the severity of the infection and the rapidity of its progress. Diagnosis in this period is difficult but important.—*L. H. Osmond.*

BÁRSONY, THEODOR, and WINKLER, KATHERINE. Fehlen der Dornfortsätze am unbegang zwischen Brust und Lendenwirbelsaule. (Absence of the spinous processes at the transition from the thoracic to the lumbar spinal column.) *Acta radiol.*, 1935, *16*, 563–570.

The authors report 9 cases in which the spinous processes in the region of the lower dorsal and upper lumbar spine were lacking. Three of the cases were in females, 6 in males. Absence of the spinous processes in this region cannot be as rare as it was formerly supposed to be as 8 of these cases were observed within a few months. In some of these cases there is also a cleft in the vertebral arch. This was the case in 4 of the authors' cases while the arch was closed in the other 5 cases. In all of the cases in which there was a cleft it was situated symmetrically in the center of the arch. In addition to the absence of the spinous processes there were other abnormalities in the development of the spine in several of the cases, such as rudimentary spinous processes, transitional vertebrae, etc. The absence of the spinous processes cannot be diagnosed clinically unless there is a defect in the soft parts manifested by a depression at the site of the missing processes. This was seen in only 2 of the authors' 9 cases.—*Audrey G. Morgan.*

FERRETTI, LUIGI. Rilievi radiologici sulle fratture delle apofisi trasverse delle vertebre lombari. (Roentgen findings in fractures of the transverse processes of the lumbar vertebrae.) *Arch. di radiol.*, Jan.–Feb., 1935, *11*, 48–59.

The clinical diagnosis of fracture of the transverse processes of the lumbar vertebrae is very difficult. The chief symptoms are intensive pain increased by movement, and inclination of the body toward the injured side. These symptoms may very readily be attributed to confusion or to sciatica, lumbago, spondylitis, etc. Roentgen examination is therefore of the greatest importance and should be made in all cases of injury of the back.

Immediately after the trauma roentgen diagnosis is comparatively easy. Fragments can generally be seen detached from the spinous processes and displaced upward or downward. Sometimes the fragment is not detached but there is a jagged fracture line. But if the examination is not made until some time after the accident the picture may be greatly changed by the callus which may be normal or exuberant, causing serious disturbances from pressure on nerves or organic changes such as inclusion of a ureter in the callus. There may be pseudarthrosis from interposition of soft parts or from fibrous callus and then diagnosis is difficult particularly if it has been a long time since the accident and the history is not very definite.

Roentgenograms are given of 5 cases from the author's practice illustrating both recent and old fractures.—*Audrey G. Morgan.*

PICCHIO, CARLO. I tumori del sacro dal punto di vista radiologico. (Tumors of the sacrum from the roentgen point of view.) *Radiol. med.*, Aug., 1935, *22*, 737–767.

The author discusses the abundant material of the National Victor Emmanuel III Institute for the Study and Treatment of Cancer. In spite of the richness of this material there were only 4 cases of primary tumor of the sacrum, 1 chordoma, 1 chondrosarcoma, 1 osteogenetic sarcoma and 1 embryonic tumor of the adenocarcinomatous type. These cases are described in detail and illustrated with roentgenograms and photomicrographs. They were characterized by osteolytic changes in the bones of the sacrum and in some cases by irregular new-formation of bone tissue.

The majority of cases of tumor of the sacrum are metastases from distant organs, particularly from the breast. The different appearances of these metastatic tumors are illustrated by roentgenograms.

Differential diagnosis is often extremely difficult. Pictures similar to those of tumor may be produced by osteoporosis, chronic osteomyelitis, Paget's disease, osteomalacia, osteopsathyrosis and tuberculous caries with abscesses and fistulas. Moreover, some pelvic metastases instead of presenting the picture of rarefaction show that of condensation. The clinical findings must always be taken into consideration in making the final diagnosis.—*Audrey G. Morgan.*

Glauner, R. Zur Ätiologie der chronischen Hüftgelenkentzündung. (The etiology of chronic inflammatory disease.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1935, *51*, 379.

The purpose of the author was to establish the relationship between chronic inflammatory disease in the female genital tract with chronic inflammation of the hip. There was such a relationship in 5 of the 20 cases he studied. These cases did not differ in any way from other forms of coxitis. Infection probably spread through the lymphatics. These cases do not always develop into arthrosis deformans despite their chronic course.—*E. T. Leddy.*

Overgaard, Kristian. Otto's disease and other forms of protrusio acetabuli. *Acta radiol.*, 1935, *16*, 390–410.

The name protrusio acetabuli is applied in the literature to different kinds of diseases. There are three chief groups of these diseases:

1. Secondary protrusions. These are complications following a clearly defined focal disease in the hip-joint or a trauma.

2. Otto's disease or osteoarthritic protrusions. This is considered a special form of deforming osteoarthritis of the hip-joint. It is generally bilateral. A certain form of the acetabulum (deep acetabulum) is thought to be the chief cause of the disease or at least a contributing factor.

3. Juvenile osteoarthritic protrusions. This disease develops at the age of puberty in women, with no signs of arthritic or traumatic changes in the hip-joint, probably as a result of weakness of the bone tissue.

The author discusses the 74 cases of the different forms of protrusio acetabuli previously described in the literature and gives brief descriptions of 13 cases of his own.—*Audrey G. Morgan.*

MacAusland, W. Russell, MacAusland, Andrew R., and Lee, Harold G. Fractures of the neck of the femur. *Surg., Gynec., & Obst.*, April, 1934, *58*, 679–698.

The authors report a series of 250 cases of fracture of the femoral neck. Only one of these was a pathological fracture secondary to carcinoma of the breast. The remainder were primary fractures and were due to tripping over a rug in the great majority of cases. They have found the vertical view of the femoral neck as described by Leonard and George to be most valuable. By means of this view, the authors have concluded that impaction of fractures of the surgical neck as so frequently reported in the past does not occur, but is due to an overlapping of the fragments as viewed in the anterposterior projection. The posterior or weaker portion of the neck crumbles, while the anterior and stronger portion of the neck, being only cracked merely buckles but is not displaced. Contrary to the belief of many surgeons, the authors believe that the proximal fragment or head is always viable. Over the end of the fragment is a heavy fibrous covering, or pannus, about which are usually attached the remains of the capsule, as well as a new fibrous tissue formation. It is the authors' opinion that the femoral head is always living and derives its nourishment through this new connective tissue.

Due to the slowness of repair, at least six months is required to obtain union in the typical case. Bone repair in the intracapsullar type of fracture takes place almost entirely by internal callus formation. The callus is too slight

in amount to be demonstrated roentgenologically. It is possible to note the progress of union about the second month; the proximal fragment shows considerable absorption, and the edge of the bone shows penciling from atrophy. Definite sharpening of the corners will be noted, causing the fractured site to have the same appearance that a sequestrum does in an osteomyelitis. By the third or fourth month new circulation has developed, a pons has grown across from the edge of the acetabulum, and new bone cells are present throughout the capital fragment. A great deal of care must be taken not to misinterpret the roentgenograms at the end of the second or third month. The marked absorption that is usually seen, with a thickening of bone on either side of a lozenge-shaped area, may be incorrectly interpreted as an ununited fracture. At this stage of healing such an appearance is of no importance, provided the fragments are still in good position. In fact, it is a stage of union, and if one persists in immobilization, the thickening begins to be absorbed, and as the shell on either side absorbs the center begins to fill in. The treatment (preferably early and accurate reduction by the Whitman method, with application of a plaster spica which is worn for six months) is described. There were good functional results in 61.5 per cent; fair in 18.7 per cent; and poor in 19.8 per cent. These results were obtained only in the recent fractures, treated early. There were 7 per cent fatalities attributed to the fractures or their sequelae.

In a late follow-up it was possible to obtain roentgenograms in 60 cases, in which firm bony union was demonstrable roentgenologically in 58.3 per cent; fibrous union in 13.3 per cent, and non-union in 28.4 per cent.—*J. H. Vastine.*

ÅKERLUND, ÅKE. Metallic scale indicators for use in nailing fractures of the neck of the femur. *Acta radiol.*, 1935, *16*, 557–562.

In extra-articular osteosynthesis of fractures of the neck of the femur with the Smith-Petersen pin the author recommends the use of two metallic scale indicators graduated in centimeters. One of the indicators is fixed to the skin with adhesive plaster in the groin, the other along the outside of the thigh in a plane which passes through the great trochanter. Roentgenograms are given showing the position of the scales; they make it possible to get an exact projection of the axis of the neck of the femur in two planes and to determine accurately the length of the nail needed.—*Audrey G. Morgan.*

WEBER, R. Über das Vorkommen von Kapselknochen bei Arthrosis deformans. (The occurrence of ossification of the joint capsule in arthrosis deformans.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, June, 1935, *51*, 541.

Two types of capsular osteoma are distinguished, the primary or synovial osteoma of Kienböck, and secondary new bone formation in the capsule resulting from arthrosis deformans and which may produce loose bodies in the joint. This paper is concerned principally with the second type. In a group of 450 cases of arthrosis deformans which the author investigated the typical picture of deforming arthrosis with secondary solitary or multiple capsule osteomas was found in 10 cases (2 per cent). According to this author any joint in the body may be involved. The knee was the most frequent site in this series—7 cases—probably because it is most often subjected to strain. The disease runs a very chronic course, and the patient may complain for years of pain and of difficulty in moving or in sudden locking of the joint. Roentgen examination shows high grade arthrosis deformans in all cases. The osteomas appear as bony tumors of various shapes and sizes. Differential diagnosis from other osseous lesions can be made only by the roentgenograms as it has a characteristic appearance unlike that of tumors of the synovial membrane, bone tumors and simple inflammatory or degenerative arthritides. Prognosis depends on the underlying lesion, deforming arthrosis. Treatment is, in general, that of the arthrosis. Operation is indicated with locking of the joint. The author illustrates some typical cases.—*E. T. Leddy.*

PRIP BUUS, C. E. Articular changes in hemophilia. *Acta radiol.*, 1935, *16*, 503–517.

Two cases are described and illustrated with roentgenograms showing the joint changes in hemophilia. The characteristic joint changes are sharply defined marginal erosions, narrowing of the interarticular space, subchondral rarefactions, rarefaction of the bone tissue unconnected with the joint surfaces, opacities in the soft tissues corresponding to the capsule, considerable destruction of the ends of the joints with resulting subluxation and in some cases changes of a more general nature, such as

calcareous atrophy and arthritis deformans. In severe cases the end result is often fibrous or osseous ankylosis, sometimes in contracted positions. Several authors state that the epiphyseal lines may be irregularly notched, wavy or have double contours. The author did not find this change in his cases. He did find a change, however, which so far as he knows has never been described before, namely, an angular bend in the jont surface, resulting finally in an abrupt right-angled break in the surface so that a part of it sinks down to a lower level.

Diagnosis of hemophiliac joint disease is sometimes very difficult. Two further cases are described in which the joint roentgenogram was that of hemophilia though the patients had no external evidence of the disease and one of them was a woman.—*Audrey G. Morgan.*

Wolke, Knut. Über Meniskus- und Gelenkknorpelverkalkungen. (Calcifications of the meniscus and joint cartilages.) *Acta radiol.*, 1935, *16*, 577–588.

The author describes 8 cases of calcification of the menisci found among 2,569 cases in which roentgenograms were taken of the knee-joints. In several of these cases there were also calcifications of the intervertebral discs and of the cartilages of the different joints. The latter findings have not been reported before. To judge from the author's cases these calcifications of the menisci and cartilages do not cause any symptoms. In 4 of his cases they persisted unchanged for from one to nine years.

The author discusses the etiology of these calcifications. He thinks they are due to decreased nutrition from senile changes in the vessels of the joints in individuals with a constitutional predisposition to changes in the cartilages.

The article is illustrated with plates showing roentgenograms of the cases.—*Audrey G. Morgan.*

Hanson, W. A., Fowler, L. H., and Bell, E. T. A case of Schüller-Christian's disease under observation for nine years. *Am. J. Cancer*, Dec., 1935, *25*, 768–771.

It is now well known that the disease is basically a disturbance of cholesterol metabolism. It is a form of xanthomatosis in which the xanthomas develop in various organs, especially the bones of the skull. The seven clinical groups of Henschen are enumerated.

The case described by the author has remained practically stationary for nine years without radiotherapy. There is involvement of the flat bones of the skull, the lumbar spine, and the ilia. An enlarged axillary lymph node removed nine years ago was mistaken for metastatic carcinoma. Attention is directed to the finding of xanthomatous lesions in lymph nodes, which may mislead the surgical patholgist.—*E. E. Downs.*

Livingston, S. K. Schüller-Christian disease (xanthomatosis). *J. Bone & Joint Surg.*, Oct., 1935, *17*, 1035–1040.

Livingston reports a cast of the rare and protracted form of xanthomatosis as manifested in an adult. In his case, the history was not absolutely typical of Schüller-Christian's disease. Frequency of urination, somewhat periodic in character, with at times passage of large amounts of urine, was present, but there had been no excessive thirst, no localized pain or tenderness. The patient did complain of headache, and also gave a history of injury to the head. The right eye was slightly prominent. Lesons were confined to the head. The blood cholesterol was very slightly raised while the blood calcium was normal.

The roentgenographic examination showed a number of areas of bone destruction in the skull, but with no definite osteoplastic reaction. Immediately above the right eye and involving the roof of the orbit, there was a defect about 13 mm. in diameter. In the left parietal area, there was a defect that measured 2 by 3.5 cm. At the vertex was another, which measured 3 cm. in diameter with two others measuring respectively, 2 and 1 cm. A linear fracture of the skull was also found.

In the differential diagnosis, the following conditions were considered and were ruled out: Paget's disease, osteoporosis circumscripta, multiple myeloma, hyperparathyroidism and metastatic osteoclastic carcinoma.—*R. S. Bromer.*

Piergrossi, Aldo. Sul tumore di Ewing. (Ewing's tumor.) *Arch. di radiol.*, 1935, *11*, 152–164.

From 1922 to 1924, Ewing described a form of tumor which he at first called endothelial myeloma and later diffuse endothelioma of bone. The histological picture has not been very clearly defined and the author discusses

the clinical and roentgen symptoms of the disease in connection with a case in a girl aged seventeen.

In 40 per cent of the cases the tumor is preceded by local trauma, the time between the trauma and the development of the tumor varying from a few days to a year. The disease occurs chiefly in males from ten to fourteen years of age, though cases have been seen in patients from forty-four to sixty years of age. In the beginning, generally before the appearance of tumefaction, the patient feels a deep pain in the limb which is often worse at night. Later a spindle-shaped swelling of the limb appears and the skin is red, hot and edematous with dilated vessels. Sometimes both pain and tumor decrease with rest and other treatment and this often leads to mistakes in diagnosis. There is often fever, usually moderate in degree. The general condition remains good for a long time; in the terminal stages there is a profound cachexia. The disease often resembles a subacute or chronic bone inflammation both clinically and roentgenologically. Roentgen treatment was only temporarily successful in the author's case. The tumor recurred and the patient died.

The author finds that there are two stages in the course of the disease. The first is predominantly osteoplastic and the second chiefly osteolytic. This is very characteristic of Ewing's tumor. As a clinical and roentgen diagnosis is not possible when the tumor is localized in the epiphyses of the long bones and in the short or flat bones, and as the histological picture is not yet very clearly defined, the author thinks the name Ewing's tumor should be applied only to tumors of the diaphyses of the long bones until its histological nature is more definitely determined.—*Audrey G. Morgan.*

MABREY, ROY E. Chordoma: A study of 150 cases. *Am. J. Cancer*, Nov., 1935, *25*, 501–517.

Chordoma is a rare and usually fatal tumor which arises from the fetal notochord. The article reviews all of the cases reported to date and adds to this number 8, making a total of 150 cases. Of this number 49 are cranial, 14 vertebral and 87 sacrococcygeal. The greatest incidence of the sacrococcygeal chordomas occurs in the fifth and sixth decades, as compared with the third and fourth of the spheno-occipital group. They occur, however, at any age, the youngest being twenty-four hours, the oldest eighty-two years. Approximately 64 per cent of the patients are male and 36 per cent female.

There are no symptoms characteristic of chordoma. Pain and evidence of tumor are the most constant symptoms. The diagnosis is difficult—a destruction of the clinoids, a defect in the clivus or evidence of intercranial pressure may be a lead in the cranial lesions. The destruction of one or more vertebrae is found in the vertebral tumors. Roentgen examination of the sacral tumors shows a soft tissue shadow with destruction of bone. The lesion is not a bone tumor and bony proliferation is not to be expected. Grossly the tumor is usually soft and jelly-like, grayish to pink in color. It is usually vascular and traversed by fibrous bands. At times it is for the most party cheesy and necrotic.

Microscopically there is wide variation. The cells have an epithelial appearance yet may be taken for cartilage. They may be separated by trabeculae and have hyperchromatic nuclei. The most characteristic feature is the appearance of vacuoles in the cytoplasm and at times in the nuclei. Spindle cells, giant cells and mitotic figures at times are found.

The prognosis is not good. Treatment is unsatisfactory. The best results are obtained by surgery. Irradiation is probably of some value in advanced cases.—*E. E. Downs.*

MENG, C. M., and CHEN, H. I. The association of intrathoracic lesions with bone and joint tuberculosis. *J. Bone & Joint Surg.*, July, 1935, *17*, 552–558.

Opinions regarding the frequency of the association of intrathoracic lesions with bone and joint tuberculosis differ. The authors' study is based upon a review of the records of 100 inpatients, treated on the orthopedic service of the Peiping Union Medical College Hospital during the past three and one-half years, of whom consecutive roentgenograms of the chest were taken.

The patients were divided into five groups according to the type of chest findings reported by the roentgenologists. Type I includes those patients reported as showing "normal chests," "lungs clear," "only pleural thickening," and "no frank tuberculosis in lungs." Type II includes those reported as showing "hilum tuberculosis" and "increased hilum with calcified

tracheobronchial glands." Type III includes those reported as "juvenile tuberculosis with involvement of adjacent parenchyma," "minimal tuberculosis," and in one case "old tuberculosis." The last three types represent definite parenchymatous lesions, active or old, minimal or extensive, and only in the first type is there no actual intrathoracic lesion.

The authors conclude that the relation between intrathoracic lesions and bone and joint tuberculosis is a close one. Of 100 patients suffering from bone and joint tuberculosis, the chest films showed pulmonary involvement in 47 per cent and intrathoracic lesions of varying degrees in 78 per cent. In view of such a high percentage, more attention should be paid to intrathoracic infections as the probable primary foci of so-called surgical tuberculosis. A multiplicity of surgical lesions, however, does not necessarily bear any direct relationship to the extent of the existing pulmonary infection. —*R. S. Bromer.*

Ghormley, Ralph K. Gas gangrene and gas infections. *J. Bone & Joint Surg.*, Oct., 1935, *17*, 907–915.

The first point brought out by the author is the fact that the idea that the diagnosis of gas bacillus infection can be made by the odor does not always hold true. He illustrates this fact by 2 cases in his experience. In the first the infecting organism resembled the *Clostridium welchii*, in the second cultures from the wound showed an organism of the *Vibrion septique* type. He tabulates 10 of a group of anaerobic spore-bearing bacilli. According to most bacteriologists, several of these organisms are often found in cultures from the same wound. Gas is produced mostly by a non-pathogenic or saprophytic organism. *Clostridium welchii* is the organism most frequently found and is one of the most pathogenic. *Vibrion septique*, or *Clostridium oedematis maligni*, while not found so frequently, is in many instances even more serious. It is important that in infections by this organism alone, the clinical picture is not at all the same as common "gas gangrene." Thus in his second case there was at no time any odor nor was there any suggestion of gas in the exudate, nor were there any crepitations.

Bacillus sporogenes is very commonly found in cultures. It is not, however, pathogenic, but is largely responsible for the putrid odor so commonly associated with these wounds. Some organisms are pathogenic, some saprophytic; thus some cases may show no odor, others may show no gas.

Thirty-three cases of gas infection occurred at the Mayo Clinic in a five year period. Causative factors were compound fractures, infection following amputations for gangrene due to thrombo-angiitis obliterans, or for gangrene due to arteriosclerosis with diabetes, spontaneous abscess in the soft parts, one case of pneumonia in which *Clostridium welchii* was isolated from the sputum, and finally, 2 cases of recovery following infection of the blood stream by the same organism, demonstrated by blood cultures.

These organisms can produce not only local changes but metastatic abscesses as well. Diagnostic aids are: (1) pain, the most common symptom; (2) swelling; (3) elevation of the pulse rate; (4) bacteriological findings, smears from the wound, cultures of the blood, etc.; (5) discoloration; (6) the presence of crepitus or gas in the exudate, not a constant sign; (7) a bad odor, also not a constant sign; (8) elevation of temperature, at times not important; (9) the present of gas bubbles in the roentgenogram of the affected part. Discoloration of the skin is at first reddish; it later becomes grayish yellow, and finally, cyanotic. The odor is putrefactive, offensive, and almost indescribable. Shadows of air bubbles in roentgenograms can also be seen in some cases of compound fracture without gas infection and also in cases of interstitial emphysema, so this sign is not pathognomonic.

Ghormley divides the treatment into four phases: (1) recognition, (2) serum therapy, (3) surgery and (4) dressings. The first is essential to proper treatment, the second is most important and the first actual therapeutic measure undertaken after the recognition of the disease. The state of progress has not yet been reached where patients with gas infection can be cured by antitoxin alone. If the extent of the infection is not enough to warrant amputation, then débridement is indicated with removal of all dead tissue. Abscesses should be drained as they appear. Widespread areas of infection should be treated with multiple incisions. If the extent of the infection is such that no doubt exists of the impossibility of saving the limb, amputation must be performed.

The dressings usually advised are potassium permanganate packs, Dakin's solution, and hydrogen peroxide. The looser the bandage, the better. Air should be permitted to reach the

wound at least part of the day. Amputations should be of the guillotine type, so that there may be no danger of closing in flaps containing the organisms.

With the judicious use of polyvalent gas gangrene antitoxin and surgery, the mortality should be reduced to approximately 15 per cent.—*R. S. Bromer.*

COOLEY, T. B., PENBERTHY, G. C., ARMSTRONG, LILLIAN, HUNSCHER, HELEN A., COPE, FRANCES, and MACY, ICIE G. Mineral metabolism; a case of osteopsathyrosis and one of ununited fracture. *Am. J. Dis. Child.*, August, 1935, *50*, 431–442.

As complete an investigation as possible was made of the mineral metabolism of 2 patients, the first, a child of eleven, female, diagnosis osteopsathyrosis; the second, a girl of seven and a half, with an ununited fracture of the tibia. The first patient had roentgen exposures made of practically the whole skeleton. These showed normal epiphyseal development and good development of the bones. There seemed to be slightly less than normal calcium content of all the bones; the decrease was most noticeable in the ribs, in which some slight cystic changes, not of the type seen in parathyroid disturbance, were observed. Trabeculation seemed to be normal. There was a marked scoliosis with no destruction of the vertebrae. The bones of the legs showed no abnormalities except slight scars of healed fractures in the left tibia and in the proximal phalanx of the fourth toe with no displacement.

The authors found some striking variations from the normal in the retention of certain elements in both children, the significance of which they could not appraise. During the five consecutive five day tests of metabolic balance, the normal child and the two children, the one with the ununited fracture and the other, the case of osteopsathyrosis showed, respectively, an average daily retention per kilogram of body weight of: 18.8, 20 and 40.6 mg. of nitrogen; 15.2, 2.5 and 2.7 mg. of phosphorus; 13.4, 8.7 and 4.5 mg. of calcium; 3.6, 2.3 and 4.1 mg. of magnesium; 32.2, 4 and −1.8 mg. of sodium; 0.4, 29.4 and 47.5 mg. of potassium, and 7.2, 3 and 4 mg. of sulphur. The control child showed a retention of 19 mg. of chlorine but for the two experimental subjects there was an extreme loss of chlorine during the first two periods of adjustment with a slight retention during the last period. So far as the clinical application of the findings in these 2 cases is concerned, the metabolic studies do not seem to justify a diagnosis of any of the classified disturbances of metabolism of bone.—*R. S. Bromer.*

INGRAHAM, NORMAN R., JR. Roentgen-positive seronegative infantile congenital syphilis. *Am. J. Dis. Child.*, Dec., 1935, *50*, 1444–1454.

This paper is an attempt to evaluate the clinical practicability, as a diagnostic procedure in early infancy, of roentgenographic study of the skeletal changes produced by congenital syphilis in the offspring of partially treated mothers. In the year from January 1 to December 31, 1934, at the Philadelphia General Hospital, even though the incidence of syphilis among the pregnant women was 11.8 per cent, and the majority of the patients received insufficient prenatal antisyphilitic therapy to insure the birth of healthy children, not one of the 1,517 babies discharged alive from the maternity ward showed any clinical evidence of congenital syphilis. The Wassermann reaction was of value in diagnosing syphilis in not more than 9 syphilitic children among 195 offspring of syphilitic mothers. The roentgenogram discovered 40 additional cases, 26 (19.4 per cent) before the age of six days and 23 cases (17.1 per cent) at ages from one to ten months. In all these cases the initial skeletal changes were evident roentgenographically before the blood serum gave a positive reaction.

Treatment of the mother prior to delivery affected the early roentgenographic evidence as follows: Of 51 cases in which the mothers were treated more than two months, in 5 (9.8 per cent) syphilis was shown roentgenographically in the infants at six days; of 68 cases in which the mothers were treated less than two months, in 21 (30.8 per cent) there was positive roentgenographic evidence of syphilis in the offspring. Cases in which the roentgenograms revealed no positive signs at the age of six days were studied at ages from three to six months. Of 36 cases adequately followed, in 12 (33 per cent) the roentgenographic evidence subsequently became positive.

In 3 cases serial roentgenograms of the same child were taken over a period of several months. These leave no reasonable doubt that the earlier bone changes seen a few days after

birth were the precursors of the more advanced and easily recognized bone lesions which developed subsequently. The Wassermann reaction which originally was negative became positive as the disease progressed, making a syphilitic etiology seem certain.—*R. S. Bromer.*

Kerr, H. Dabney, and Berger, Raymond A. Bone metastasis in carcinoma of the stomach. *Am. J. Cancer*, Nov., 1935, *25*, 518–529.

One hundred and forty-three cases of carcinoma of the stomach in which bone metastases were found were reviewed and to this group the authors added 5 cases. From their study they concluded that metastasis to bone was more frequent at the sites of red bone marrow—spine, ribs, femur, sternum, and pelvis. Both osteoplastic and osteoclastic lesions were found. The site, size, and type of the primary tumor had no influence on the appearance of the osseous involvement. Bone metastases were more frequent in the relatively young patient. Dissemination was probably through the blood stream. The anemia found in a number of cases could not be distinguished morphologically from a primary type.—*E. E. Downs.*

Keyes, E. Lawrence. Anatomical observations on senile changes in the shoulder. *J. Bone & Joint Surg.*, Oct., 1935, *17*, 953–960.

Keyes examined the shoulder joints of 98 cadavers, bilateral observations being made in all but 4 instances. Certain lesions of the joint were found, listed as follows according to frequency of occurrence:

1. Partial rupture of the tendon of the supraspinatous muscle.
2. Knobbing or irregular hypertrophic changes of the greater tubercle and adjacent regions of the humerus.
3. Deposits of calcium within the supraspinatous tendon.
4. Adhesions within the subacromial or subdeltoid bursa.
5. Erosion of the floor of the subacromial bursa.
6. Increased fragility of the bone of the greater tubercle.
7. Erosion of the articular surface of the humerus.

Keyes lists certain negative findings:

1. In no instance was the tendon of the supraspinatous muscle found completely torn through—from border to border.
2. No tear of any tendon other than the supraspinatous was found.
3. The long head of the biceps brachii muscle was intact in every instance. Fraying of the tendon was not observed.
4. The head of the humerus was dislocated in no instance.
5. Evidences of old or recent fracture of the humerus, acromion, or clavicle were lacking.
6. Erosion of the articular surfaces of the shoulder joint, unassociated with rupture of the tendon, was not observed.

Partial rupture of the supraspinatous tendon was the most frequent lesion found and occurred more often in the cadavers of older ages. Keyes concludes that these lesions in the shoulder are to be considered traumatic and degenerative in nature. Age, long use, and trauma seemed to be the chief causative factors. Alterations in tissue growth and metabolism, he believes, act locally to aid the formation of the lesions. Infection was thought to be the cause in only a very few. The article is illustrated with microphotographs of the torn tendons and calcifications, etc.—*R. S. Bromer.*

Zeitlin, A. The traumatic origin of accessory bones at the elbow. *J. Bone & Joint Surg.*, Oct., 1935, *17*, 933–938.

Accessory bones may be divided into two groups. The first type may be encountered most often near the carpal and tarsal joints and is regarded as a normal variation. The second group includes rare bone structures at other joints. In the cases reported in the literature, the elbow joint is most frequently involved and the supernumerary bones are found in three typical locations: distal to the medial humeral epicondyle, proximal to the olecranon, and near the trochlear fossa. The question of traumatic or congenital origin is always raised.

Zeitlin reports 3 cases: the first showed on roentgen examination, an oval bone shadow, 1 by 1.5 cm., with well-defined cortex and spongiosa, situated 5 mm. from the medial humeral epicondyle. The second showed a small round shadow, the size of a hazelnut in a similar position to that found in the first case. The third showed a supernumerary, isolated, sharply demarcated, elongated bone with a distinct spongiosa situated at the tip of the olecranon.

In all of these cases, Zeitlin concludes that

he was not dealing with a recent avulsion as a cause. He does not believe in their congenital origin. He agrees with the theory that these bodies are the result of traumatic avulsion of the ossification center in early childhood. This is the cause of the round epicondylar bodies in the opinion of some authors, and of the cubital patellae (third case), according to Kienböck, Katz and Gunn. Zeitlin has collected a number of roentgenograms of traumatically displaced epicondylar apophyses in children of different ages and these show the displaced fragments to be separated from the condyles by a distance of a few millimeters up to 1.5 cm. Check-up roentgenograms after a few months show definite evidence of concentric growth of the fragments with gradual assumption of the rounded form. All reported cases show the surfaces of the medial epicondyle and the neighboring part of the humerus, smooth and with no definite evidence of recent trauma. There is no loss of function of the joint. These isolated ossicles are often accidental findings. There are other causes of new bone formation, such as arthrosis deformans and neurotrophic arthropathies but in these cases the underlying disease is so typical and the roentgen appearance so definite that there is no doubt of the diagnosis.—*R. S. Bromer.*

AITKEN, ALEXANDER P. Further observations on the fractured distal radial epiphysis. *J. Bone & Joint Surg.*, Oct., 1935, *17*, 922–927.

This paper is an elaboration of a previous report, Aitken having collected and observed further cases of fracture and dislocation of the distal radial epiphysis. Two of his conclusions in his previous study are confirmed, namely, regardless of the amount of displacement of the center, the epiphysis returns to normal position within a period of two to three years after the injury, and second, although deformities following such injuries are seen at times, in the larger hospitals, the actual occurrence of deformity is relatively rare in proportion to the total number of injuries. These deformities are due to crushing injuries of the epiphysis and are in no way attributable to malposition.

Reduction is accomplished by the production of bone on the dorsum of the shaft, so that the shaft is brought up to the epiphysis. The volar portion of the shaft undergoes absorption. The loss of the volar bowing is restored within two years. Temporary retardation of growth is commonly noticed, but is of no clinical importance. Reduction occurs at any age, regardless of the proximity to the normal ossification center. His one case of deformity in the series he regards as due, without doubt, to crushing of the epiphysis which was demonstrated by the roentgenogram.—*R. S. Bromer.*

MASERITZ, I. H. Acute intermetacarpophalangeal calcification. *J. Bone & Joint Surg.*, Oct., 1935, *17*, 1017–1020.

Maseritz reports a case of acute inflammation with calcification, involving an intermetacarpophalangeal space of the hand. The clinical findings pointed to a tendonitis of the interosseous muscle or to a possible acute calcified metacarpophalangeal bursitis. In the absence of operative intervention he felt that the changes, for which he can find no description in the literature, could have been only those of an acute intermetacarpophalangeal calcification.

The roentgenograms showed calcifications in the intermetacarpophalangeal space between the index and middle fingers, right hand. Later these were almost completely absorbed. Associated with this was definite calcification of the subdeltoid bursa of the same side. Between the interossei muscles lie the metacarpophalangeal bursae which are said to extend from the metacarpal heads to the bases of the first phalanges of the corresponding spaces and are related laterally to the interossei muscles and, ventrally, to the transverse ligaments. Maseritz feels that in his case, one cannot dismiss the possibility of a calcified bursitis. Pathological conditions such as myositis ossificans, Raynaud's disease, calcinosis interstitialis and other like processes could be ruled out.—*R. S. Bromer.*